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MEDICAL AND SURGICAL REPORTS

OF

THE BOSTON CITY HOSPITAL.

Sixth Series.

EDITED BY

GEORGE B. SHATTUCK, M.D., W. T. COUNCILMAN, M.D.,
AND H. L. BURRELL, M.D.

BOSTON:
PUBLISHED BY THE TRUSTEES.
1895.

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BOSTON.

PREFACE.

The Sixth Series of the Medical and Surgical Reports of the Boston City Hospital follows the Fifth Series at the interval of a year, and it is hoped that the publication may in future be an annual. The last series marked the completion of the thirtieth year of the Hospital. The last year has shown the usual steady growth of the Hospital in size and efficiency, and beyond that there have been few changes. Another year it is probable that the editors may be able to announce the completion of several new and important buildings now in process of construction, and to publish some account of their internal arrangements.

Future series should contain valuable additions to the scientific knowledge of some of the infectious diseases. The editors are glad to be able to present in this series a report from a Committee of the Visiting Staff, upon "Infectious Diseases, and the Management of Hospitals devoted to Infectious Diseases."

Since the publication of the last volume, the Visiting Staff has lost by resignation the much-valued association of Doctor E. H. Bradford.

The Editors.

May, 1895.

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I.

SUDDEN DEATH BY THE RUPTURE OF THORACIC ANEURISMS PREVIOUSLY UNRECOGNIZED.

By F. W. Draper, M.D.

The autopsy-room of a hospital is specifically dedicated to the study of morbid anatomy. Its air is heavy with the effluvia of disease, decay and death. Its ministering spirits, with erudite touch and penetrating vision, search out the secrets of nature. Infection, inflammation and degeneration render here their final account. The pathologist supplies the closing chapter in the natural history of diseases whose earlier incidents are written in the clinical records relating to the living patient. The first and the last observations upon the human subject, in connection with his fatal illness, sometimes fail to connect harmoniously, and the ward bed and the marble slab sometimes present details which do not blend. Nevertheless, the aid which clinical medicine derives from the anatomical amphitheatre of the hospital is too obvious to need elucidation. The aphorism of Dr. Holmes, that while pathological anatomy teaches a great deal, "it is, after all, like inspecting what is left of the fireworks on the morning of the fifth of July," is less true to-day than when he wrote it. Histology and bacteriology now lend their effective aid to the grosser methods of observation, and pathology coöperates heartily with clinical medicine in the advancement of medical science.

But the autopsy-room of the City Hospital of Boston has not exhausted its usefulness, great as that service is, by discovering and recording the anatomical appearances which the hospital patients bring to the dead-house after a period of observation in the wards. Through the courtesy of the trustees, and with the indulgent and cordial assent of the

pathological department, autopsies are made here upon the bodies of such victims of crime or such subjects of sudden and suspicious deaths in the southerly portion of Suffolk county as require an official medical examination. It thus happens that many interesting medico-legal problems are presented here for solution. The privileges bestowed upon the medical examiner by the hospital place under him continued obligation. It is in recognition of this debt and of the very great help in the medical examiner's work which it implies that he accepts the invitation to contribute to these pages.

In fulfilment of this design, I have selected from notes of autopsies performed by me at the hospital a series of cases of thoracic aneurism. These cases are all instances of sudden death. Their subjects have this in common, that the disease of the aorta which ended life so suddenly had not been suspected or recognized or been made the occasion of medical observation or treatment. In this respect they illustrate a rule about which all writers are agreed, that a large number of aneurisms declare no sign of their existence, and that they are revealed by the post-mortem examination only. They show how imperceptible and with how little injury to health may be the growth of even a large aneurismal tumor. Finally, they are examples of the usefulness of autopsies in determining accurately the causes of tragically sudden death, and in taking the obscurity out of cases which are too often dismissed with a convenient expression like "heart disease," or the more objectionable but equally convenient phrase "heart failure."

I. Josephine S., aged 37, a hard-working charwoman, entirely free from disabling disease though a moderate user of alcoholic drinks, was found dead, lying in a natural attitude on the outside of her bed, with her outer clothing removed. She was last seen alive twenty-three hours previously. Her body when discovered was cold, and rigor mortis was fully developed. Her acquaintances supposed her to be a healthy woman.

Autopsy. — The body was spare but not emaciated. The

countenance presented no suggestion of a painful death. The surface of the body was pale.

When the sternum was lifted the left pleural cavity was found filled to its fullest capacity with yellow serum and dark-red, firm blood-clot, the whole mass measuring two quarts. This blood had escaped from a rupture in the inferior wall of an aneurism of the aortic arch. The aneurismal tumor projected posteriorly opposite the fourth, fifth and sixth dorsal vertebræ, and had eroded the vertebral bodies and intervertebral cartilages in this region to the depth of a quarter of an inch at the deepest, leaving the general surface roughened and irregular. The aneurismal sac was ovoid in shape, and measured four inches long, three inches wide, and one inch and a half thick. Within it were masses of firm coagulum, with dry, shreddy layers of fibrin.

The rupture was at the lowest part of the sac. The opening was valvular, from above downward. Externally it appeared as a rent in the aneurismal wall just below the root of the left lung, the lesion being irregularly crescentic, with the concavity downward, and with an opening at its uppermost part admitting a large probe.

The heart was normal. The left lung was compressed, dry and pale; the right lung was of normal volume, and moderately reddened. The other organs showed nothing noteworthy.

II. John J., aged 43, intemperate and shiftless, had not been heard to complain of any ailment until the morning of the day of his death. At this time he procured from a druggist a "plaster for his chest." About three hours later he was seen on the street, looking so distressed that an acquaintance remarked upon his appearance, and J. replied, "I *am* sick." Presently he went into a store and sat down; he quickly became stiffened in a convulsion, was rigid and unconscious, breathed stertorously, gasped a few times, and died, the whole transaction occupying but a few moments.

Autopsy. — The surface of the body was very pale; there were scarcely any lividities of the skin. The mucous surfaces were blanched. Cadaveric rigidity was pronounced.

The heart was healthy and empty. The lungs were normal, though rather anemic.

In the abdominal cavity were sixty-six fluid-ounces of mingled fluid blood and blood-clot. The source and course of this hemorrhage were found to be as follows: At the junction of the transverse and descending portions of the aortic arch there was a projecting aneurism, with thin walls, the tumor being of the size of an English walnut. This outgrowing sac had ruptured at its outer part, and the escaping blood had dissected its way inside the sheath of the aorta, leaving a blood-stained track in the posterior mediastinum. It had then torn through the diaphragm between the aorta and the œsophagus. It had then turned to the right, across the cardiac end of the stomach posteriorly, and here tearing away the peritoneum of the stomach over a surface two inches by three inches, it had broken through and discharged into the general peritoneal cavity.

All the organs of the abdomen were structurally healthy.

The brain was pale and wet.

III. Winifred M., aged 65, a homeless wanderer, depending on street alms and the poor hospitality of her acquaintances to keep her from starvation, was not known to be the subject of any disease, although she had been heard once to make an allusion to her "heart" as the seat of some indefinite disorder. On the day before her death she was on the street from morning till night, and returned to her temporary lodgings in her usual health. Her bed was prepared for her on the floor, and she went to sleep at ten o'clock. In the night she shifted her bed from the corner of the room to the middle; and here, at 7 A.M., she was found dead without any indication of distress in her attitude or countenance.

Autopsy. — The body was emaciated. The surface was pale, with some cadaveric lividities upon the dependent parts.

When the sternum was removed, the pericardium was observed to be distended, bulging outward, and freely exposed; it contained ten fluid-ounces of blood, fluid and clotted. The heart was moderately and symmetrically enlarged; its

muscle was readily crushed by finger pressure. The aortic valves were moderately thickened at their free border, but were competent. The mitral valve was normal.

The aorta, beginning at a point near its origin, was dilated; the enlargement was rather greater on the upper or convex border, was fusiform in shape, without sacculation, and in size and contour resembled a lemon. The wall of the dilated artery was thinned to an appreciable degree; and the intima, not only of the arch, but of the entire thoracic portion as well, showed large patches of atheroma, some of them with calcareous plates.

Immediately to the right of the innominate artery, on the upper curve of the transverse part of the arch, there was a rent in the intima of the aortic wall, transverse in direction, and one inch long; the edges of the rupture were irregularly indented, but the general direction was linear and straight. The situation of this internal rent was above the line of the pericardial attachment. But the blood, instead of forcing its way directly out at this point, had dissected a passage backwards and downwards, along beneath the outer coat of the artery, and had found its entrance into the pericardial enclosure, through a longitudinal rent an inch and a half long, and two inches below its rent of exit through the inner coat of the aorta. The tissues between the two ruptures were infiltrated with blood.

Both pleural cavities were obliterated by old pleuritic adhesions. Both lungs were engorged. There was no blood in the bronchi.

There was no sign of disease about the abdominal or pelvic organs. The brain was normal in appearance.

IV. Joseph F., aged 75, was alive and well at 6.30 A.M., when his room-mate left him at home. He had been out of doors through the previous day, and was not known to have any ailment. At 1 P.M. he was found dead on the floor of his bedroom. He was in a kneeling posture, with his forehead resting on the floor in front of him. His trousers were down about his thighs.

Autopsy. — The body was spare. Rigor mortis was fully developed. The face was livid.

The pericardium was distended and bulging, showing a dull purple color through its wall. It contained twelve and a half fluid-ounces of blood, fluid and clotted, the clot being soft and of a dark color.

The heart was normal in appearance; its valves were all competent and in healthy condition.

The aorta was dilated: its intima was thickened and roughened from the semi-lunar valves to the iliac bifurcation, the result of atheromatous degeneration.

Just above the anterior coronary orifice, in the ascending portion of the aortic arch, there was an aneurismal sac projecting outward and to the right, and having a cavity of about the size and shape of a large olive. At the outer aspect of this small sac was a crescentic, valve-like rent, extending downward into the pericardium. The external lesion in the outer coat of the vessel was curvilinear, three-quarters of an inch long. The orifice through which the blood escaped through the intima was irregularly rounded and of about the size of a pea.

The lungs were injected. The spleen was softened almost to a condition of diffluence; it was small, dark-red, and adherent to the adjacent tissues by the products of former inflammation. The stomach and intestines were normal. The liver showed the appearances of fatty infiltration. The kidneys represented chronic interstitial inflammation. The brain was unnaturally moist.

V. Leodegar M. L., aged 51, was found dead in his room in a hotel, lying back against some pillows on a lounge, one foot on the lounge and the other hanging off its edge. An hour and a half previously he had walked up the three flights of stairs leading to his room, and seemed in his usual good health and spirits when he entered the hotel office. He was the subject of a lateral curvature of the spine, and to his acquaintances this appeared to be his only disability. It was ascertained from one of his intimate friends, however, that for many months he had had pain in his shoulders and in the right sub-clavicular region, to such a degree that he had talked of sudden death as a possible termination of his trouble. The last evening of his life he had seen a well-

known and experienced physician who gave him some advice of a general nature, but did not entertain the idea of any serious organic disease.

Autopsy. — The body was emaciated. The countenance indicated distress. There was a marked lateral curvature of the spine in the dorsal region, which gave also a "pigeon-breasted" deformity to the sternal region.

When the sternum was removed, the pericardium was observed to be prominent, distended and elastic, showing dark contents through its translucent wall. It contained sixteen fluid-ounces of blood, fluid and clotted. The source of this hemorrhage was found to be a minute rupture in the wall of an aneurism of the ascending and transverse portions of the aortic arch. This portion of the artery was enlarged to about twice its normal size; its intima was rough and thick. From the outer and upper part of the ascending section a secondary projection was found, the result probably of a former rupture of the aortic wall. The wall of this smaller sac was relatively thin, and was lined with loose, rather dry clot. At its upper border, as close as possible to the pericardial line, was a minute orifice, hardly admitting the head of a small probe. The size of the sac was that of a large apricot.

The heart was firmly contracted and empty: its structure was normal. There was no effusion of blood outside the pericardium. The lungs were injected; in other respects they were normal. The spleen and kidneys were reddened. The other organs were healthy in appearance.

VI. John H. H., aged 51, ran to catch a train which was about to start. He entered a car and took his seat. Presently he was observed to be ejecting blood copiously from his mouth; his death quickly followed. It was learned from his kindred that he had been ailing many months with some obscure symptoms which represented "heart disease" to him; but he had not been incapacitated for the performance of his daily duties. He himself had anticipated sudden death.

Autopsy. — The body was sparely nourished. The surface was pale. Post-mortem rigidity was pronounced.

Dried blood was in the nostrils, on the moustache and beard, and on the lips and teeth.

On raising the left lung, a firm adhesion was found posteriorly, near the apex, tying the lung to the spinal column and the adjacent ribs, over a surface two inches in diameter. When the heart, lungs, œsophagus and aorta were removed together, this area of adhesion was found to be a portion of the wall of an aneurism of the thoracic aorta, involving the lower section of the descending portion of the arch as well. The aneurism consisted almost wholly of the dilated arterial walls, much thinned; there was no deposit of fibrinous layers within it. The lining surface was rough from atheroma, with projecting calcareous plates. The part next to the spine, where the adhesion was found, had eroded the bodies of the vertebræ to a slight extent. Just in front of this eroded space, the aneurism had ruptured in a forward and upward direction into the upper lobe of the left lung. At the point of rupture, the wall was thinner than elsewhere. The rent was irregular and somewhat stellate in shape, and admitted the tip of the finger. The size of the aneurismal cavity was about that of an orange.

The entire arch of the aorta was considerably dilated, and its intima was roughened and thickened. The aorta below the aneurism also showed atheromatous changes, without dilatation.

The heart showed no change whatever from the normal condition, in size, thickness of its walls, color of its muscle, or state of its valves. Its cavities were empty, and its left ventricle was contracted.

The left lung at the point opposite the aneurismal rupture showed an excavation half an inch deep and of irregular periphery; all around this the tissues were infiltrated with blood, giving an engorged appearance to the entire upper lobe, and, to some extent, the upper part of the lower lobe also. There was no blood in either pleural cavity. The right lung was somewhat reddened. The trachea and bronchi contained blood, and there were about four fluid-ounces of clotted blood in the stomach.

The other organs showed nothing noteworthy.

VII. An unknown woman about 60 years old, presenting the aspect of a homeless street-beggar, was seen to stagger across Atlantic avenue and to drop helplessly upon some store-steps. She was bleeding from the mouth. She was insensible when the by-standers reached her, and presently she died. The front of her dress was blood-stained, but the evidences of very profuse hemorrhage were wanting. The body was not identified, and therefore all knowledge of her health previous to her attack on the street is lacking.

Autopsy. — The following appearances were the only ones of immediate interest: The upper portion of the thoracic aorta, just below the arch, presented an aneurismal dilatation of the size of a large orange; it was located opposite the bifurcation of the bronchi. Portions of the wall were very thin, while other parts were much reinforced by layers of fibrin. The aneurism had ruptured into the bronchi. There was no blood in the pleural cavities. The lungs were engorged, and the bronchi and trachea contained frothy blood. The heart was enlarged, the hypertrophy being chiefly on the left side. The aortic valves were thickened, and behind them were small calcareous plates of great density. The aorta was dilated just above the valves, and at one point in the ascending portion the walls had yielded, forming a depression one inch in diameter and a quarter of an inch deep. The intima of the aorta throughout its extent was thick and rough.

VIII. Michael F., aged 77, was seen to topple over as he was in the act of crossing Park square at 10 o'clock A.M. Medical attendance was immediate, and the man was placed in an ambulance and transferred quickly to the City Hospital. On his arrival, he was in a state of profound collapse. He gave his name and address, but no other facts. He died at 10.45 A.M., three-quarters of an hour after the seizure. His family stated later that they had had no knowledge of any disability of which he was the subject.

Autopsy. — The body was spare, and its surface was pale and exsanguine. The pericardium, prominent and bulging when first exposed, was found to contain a pint and a half of fluid blood and soft clots. The heart was moderately

enlarged, and its left ventricle showed a thickened wall; its valves were normal.

The arch of the aorta was much dilated, its lumen being expanded into a cavity of the size of a large lemon. In the descending portion of the arch, just behind the pulmonary artery, there was a transverse rent in the intima, two and a half inches long, linear, straight, but not penetrating into the pericardium; the most careful exploration failed to discover any communicating opening, and the conclusion was that the leakage seemed to be by filtration through the outer layer of the aorta's wall. The inner coat of the aorta was thick and rough, and the lumen of the artery below the arch was narrow and irregular. The tissues in the posterior mediastinum and in the posterior parts of both lungs were fully infiltrated with blood; the lungs elsewhere were œdematous. About a pint of thin, bloody serum was found in the pleural cavities.

The spleen, pancreas, stomach and intestines were normal. The liver represented passive congestion. The kidneys were typical specimens of chronic interstitial inflammation.

IX. Chipman H. S., aged 37, was admitted to the City Hospital about 10 o'clock P.M., January 20, at the request of a physician who stated that the man was suffering from paraplegia, in some way connected with two doses of a poisonous drug prescribed by an irregular practitioner. It appeared from S.'s story that he had not suffered any disability until the forenoon of that day; at this time his "legs gave out" and he sank to the floor of his room. He said he had great pain in his back, not localized, but general. There was also an account of vomiting and of the passage of bloody matter in his dejections, and this lent some aid to the theory of poisoning.

At the hospital a loud souffle was heard over the heart. The man was restless, very thirsty, and disinclined to talk. Toward morning, January 21, he sank into a stupor and had some equivocal convulsive movements. He gradually failed, and died at 12.05 P.M. that day. The hospital diagnosis was not conclusive, but cerebral hemorrhage seemed the probable condition.

Autopsy. — The body was well nourished and of good muscular development. Cadaveric lividities were more than ordinarily pronounced on the lateral and dependent parts.

The heart was hypertrophied without dilatation. Its valves were all in normal condition. The myocardium was pale but firm. The aortic arch was dilated moderately and displayed old endarteritis. The aorta below the arch and to within five inches of the diaphragm was normal. At this point, just above the diaphragm, there was an aneurism of the size and shape of a large kidney. Its anterior wall was adherent to the posterior surface of the lower lobe of the left lung. Its posterior wall was the exposed and eroded bodies of the eighth, ninth and tenth dorsal vertebræ; the necrosis of these bones had gone so far as to leave upon the front and left side deep excavations into which the forefinger could easily be laid, leaving a thin shell of denser bone at the upper and lower margins, with the intervening cartilages in place. Around this bony destruction the aneurism was firmly attached to the spine at either side. The contents of the sac consisted of laminated and rather dry, easily broken blood-clots, brownish-red in color, through which ran a tortuous blood canal of the size of the finger.

The lungs, spleen, stomach, pancreas, intestines and liver were healthy. The kidneys were injected. The brain was of normal appearance.

The spinal cord was taken out from behind. The vessels of the dura were injected throughout the dorsal region. The body of the tenth dorsal vertebra, in front of the dura, was carious and dark-red. The ninth vertebra showed less change. There was a marked excess in the spinal fluid, fully distending the cavity of the dura. The vessels of the pia were fully injected, especially along the anterior surface of the cord. In the anterior and left lateral columns of the cord, near the affected vertebræ, there was a marked degree of reddening, in contrast with the color above and below; but the consistence of the cord was normally firm.

X. Patrick J. S., aged 30, a tailor by trade, had been ailing many months with an obscure pain in his right side; and also, at times, with what he had called "neuralgia of

the heart." He had received medical treatment for "liver complaint." He had kept at his work, without interruption, up to the hour of his death. The first symptoms of his approaching end were pallor and great dyspnœa. He entered a liquor store, where he was known, and sat down "panting for breath." A carriage was called, and the man was placed in it and started for the City Hospital. He died on the way.

Autopsy. — The body was lean. There was rather more than the usual degree of cadaveric lividity.

The left pleural cavity contained ten fluid-ounces of thin, red fluid. Both lungs were, in general, gray in color, and were thoroughly œdematous and sodden. The right lower lobe and left upper lobe were injected more than other parts; but the reddening was very moderate in degree. The pericardium contained five fluid-ounces of straw-colored serum. The heart was normal in size and color. Both ventricles were firmly contracted; their walls were not thickened. The several valves were normal.

An aneurism projected from the anterior wall of the aorta, immediately above the semi-lunar valves. Its size was that of a small orange. Its wall was translucent at its most projecting part; elsewhere it was lined with friable coagula, or masses of fibrin, and was opaque and thickened. The orifice of entrance to the sac, seen from within the aorta, was one inch above the right semi-lunar valve, and appeared as a round aperture through the wall of the vessel; the opening admitted the tip of the forefinger.

The aneurism was so located that it overlay and nearly obliterated the lumen of the pulmonary artery just above its origin at the heart. The aortic arch was moderately dilated, and its intima was thick and uneven.

The other organs of the body displayed nothing noteworthy.

In addition to the features which the foregoing cases have in common (the suddenness of their termination and the fact that the development of serious aortic disease had never been discovered or treated), these deaths suggest some points of special interest, among which the following may be deemed worthy of mention:

1. The rupture of an aneurism of the aorta, although it sometimes has physical exertion as its exciting cause [Cases V. and VI.], does not of necessity require such a cause [Cases I., III., VIII.].

2. The pericardium, more often than other cavities, receives the escaping blood; and, in these cases, it is not the amount of the hemorrhage which kills, but the inhibition of the heart's action, by compression of the cardiac walls, in a sac filled to distension with blood [Cases III., IV., V., and VIII.].

3. Deaths by aneurismal rupture, although deserving the description "sudden," are not instantaneous; an appreciable interval [Case VI.], and, sometimes, a very considerable period, elapses between the attack and its termination [Cases VII. and VIII.].

4. A death by aneurismal rupture is not generally a painful one [Cases I., III.]; although it may leave evidences that it is so in some cases [Cases IV. and V.].

5. The escaping blood in an aneurismal rupture does not always follow the line of least resistance [Case II.]; and, sometimes, it makes a devious dissection to reach an outlet [Cases III., IV.].

6. Its small size and fusiform shape do not give an aneurism immunity from sudden rupture [Cases II., III., IV., VIII.].

7. An aortic aneurism is not necessarily an affair of advanced life [Cases I., II., V., and VI.].

II.

OBSERVATIONS ON THE DIAGNOSIS OF DIPHTHERIA AND A STUDY OF ITS LOCAL TREATMENT.

By Francis H. Williams, M.D.

I shall assume that diphtheria is caused by the Klebs-Lœffler bacillus and begins as a local disease. The diphtheria bacillus flourishes chiefly in the throat and is not a motile bacillus, but must be carried to the healthy individual. This is done principally by direct transferrence from mouth to mouth, or hand to mouth or nose. By mouth to mouth I mean, for example, a healthy child may catch diphtheria by biting from the same apple, putting the same toys in the mouth, using the same handkerchief as a child in whose throat diphtheria bacilli are present, or by kissing such a child; or a mother may use her own handkerchief for a neighbor's ailing child, and thus unwittingly carry the disease to her own children. The closer contact among children, and the habit they have of putting things in their mouths, shows why the disease is more prevalent among them than among adults. The bacilli may get on to the hands of those who are taking care of or are about persons ill with diphtheria, and unless the hands are carefully washed may be transferred to the mouth at the next meal on a piece of bread, for instance. Again the bacilli may live for months in the garments of a person who has been ill with diphtheria. But I need not multiply examples as to the mode of contagion.

The bacilli, once conveyed to the mouth or nose, may remain in the throat some days, or more than a week even, without showing any sign of their presence, and in some cases may produce no symptoms of disease, although during this

period such individuals may give diphtheria to others; but in the usual course of things the presence of bacilli in the throat is followed, probably within a day or two, by congestion, although this congestion may not be marked, and usually the tonsils, palate, or pharynx are first attacked; this congestion is sometimes accompanied by much œdema and swelling, and is generally soon followed by the appearance of false membrane, which may at first cover a small area only, but following the spread of the congestion may appear in new areas on each day for a few days; in other words, the mucous membrane is, as it were, planted with bacilli, and the false membrane results, but the crops may not all appear at once. Most of the bacilli are found in the outer portions of the false membrane. In a word, the natural history of the disease seems to be: bacilli on a most alkaline surface, at a temperature favorable to their growth, irritation, congestion, false membrane. This membrane may disappear from the throat before the bacilli. Therefore in the beginning and at the close of the disease an individual is especially dangerous to the community, because in the first case his diseased condition is unsuspected or thought of slight consequence, and in the second case because the normal appearance of the throat, although the bacilli may still be present, leads to the belief that the danger of contagion from him is past. The bacterial test, therefore, is of special importance in these two stages of the disease, as it is the only means of detecting the diphtheria bacilli.

In 1892 I began to employ the bacterial test for my patients and initiated its use at the Boston City Hospital. Since the beginning of 1893 this test has been carried out without interruption in the pathological laboratory of the hospital, under the direction of Prof. W. T. Councilman, and it has now been applied to a large number of cases, among them to more than 500 of my patients. A number of bacterial examinations have also been made for my patients in the laboratory of the Harvard Medical School.

As already stated, there are two stages of diphtheria in which this test is especially serviceable.

First. Very early in the disease, before the throat shows any trace of false membrane.

Second. Late in the disease, after the false membrane has disappeared; that is, during convalescence.

Let me illustrate the *first class* of cases by giving the history of a household in which a case of diphtheria occurred. A patient of Dr. James J. Minot, a boy of eight years, had sore throat with membrane on a certain Sunday; bacterial examination showed on Monday evening that the bacilli of diphtheria were present. Dr. Minot then put the patient under my care. The throats of the other members of the household presented no signs of diphtheria, but it seemed to me wise to follow my rule and have swabs taken from the throats of all. They were also taken by Dr. Minot from the throats of those outside of the household who had been much with the patient. In three of the members of the household bacilli of diphtheria were found, although, as already stated, there was nothing to indicate diphtheria to the unaided eye when the swabs were taken for diagnosis. One of these three throats was carefully examined with the mirror by a laryngologist shortly after the swab was taken and he found no sign of diphtheria. Thirty hours later there was no indication of membrane in two of these throats. Treatment with strong hydrogen-dioxide acid solution was begun on all these cases as soon as the Klebs-Lœffler bacilli were found, and in one of the adults the dioxide made conspicuous the false membrane on the right tonsil and anterior pillar which was barely visible just before its application. No membrane was seen at any time in the throat of the second patient, a boy of two years. After three days' treatment with strong hydrogen-dioxide solution the bacilli of diphtheria were not found in cultures from his throat. The third member of the household referred to above, the nursery-maid of the first patient, had no sign of false membrane in her throat twenty-four hours after the swab was taken, but within a day or two it spread to nearly every part of the throat within sight, or which could be seen by depressing the tongue strongly. The diagnosis of diphtheria could not have been made in these cases, except by these bacterial examinations, at the stage of the disease when the swabs were taken. These four cases all recovered.

The advantages of early diagnosis, of early isolation, and

early treatment are seen by contrasting the above-mentioned household with the following family:

In this family, a child was taken ill with diphtheria, a few days later a second child, who died soon after at home, and at short intervals two other children, the mother and aunt. Three children, the mother, and aunt came to the Boston City Hospital and were under my care. The children came too late to be helped, and all died. The mother and aunt were the last members of the family to show signs of the disease; they entered the hospital when they had been ill three and four days respectively, and recovered.

Such experiences show that as soon as a case of diphtheria is found in a household, the bacterial test should be applied to all its members, though their throats may be normal in appearance. This would lessen the spread of the disease and give more patients the great advantage of early treatment; in some cases this might be begun before any other warning had been given than the presence of the bacilli.

In the *second class* of cases — that is, during what I may call convalescence — we frequently find that although the throat of a given patient looks perfectly well, yet it still contains bacilli, and isolation has therefore to be continued. The safeguard which bacterial examinations of the throats during the convalescent stage may be to the community is well illustrated by one of my patients (treated locally with corrosive sublimate 1 to 10,000, and a weak (10 volume) solution of hydrogen dioxide), from whose throat the false membrane had disappeared and who had expressed a hope to be at home for the Thanksgiving festival. Formerly, discharge being determined by the inspection of the throat only, she would have been allowed to leave the hospital much before the desired date. The bacterial test, however, which was made at short intervals for some weeks after the date of the festival referred to above, showed the bacilli to be present. One shudders to think of what might have followed her contact with the children of the family, and how, originating with her, the disease might have spread to other families.

In a former paper[1] I have referred to the persistence of

[1] "American Journal of the Medical Sciences," November, 1893.

the bacilli in the throat for some weeks, and in one patient (treated locally with corrosive sublimate 1 to 10,000) for nearly three months, and pointed out the necessity of making a bacterial examination of the throats of patients before isolation is terminated.

We now come to another stage of the disease in which there are acute appearances in the throat, with false membrane. In these cases the Klebs-Lœffler bacilli are usually found. We may find cases, though they are uncommon, in which one, two, or even three examinations fail to find the bacilli of diphtheria, although direct examination of the membrane itself sometimes shows them to be present. Further, we may have cases in which no bacilli are found in the first examination, but subsequent trials show them to be present; and, finally, we may have cases in which the bacterial examination during life fails to find them, although a post-mortem examination reveals them. On the other hand, unless the test is carefully made it is quite possible to mistake some other bacillus for the bacillus of diphtheria. I therefore feel called upon to repeat my statement of last year, that these examinations are not infallible; but when all precautions have been carefully taken, and more than one examination made, I believe that they may usually be relied upon, except in cases where the disease is laryngeal, and that we are certainly justified in taking a much more favorable view of the recovery of a patient in whose throat the bacilli are not found than in those where they are found, although the appearances in the throat may be similar in both. Failure to find the Klebs-Lœffler bacilli may mean that they have perhaps become less abundant through the more vigorous growth of other and less dangerous organisms. It may be that this is one of nature's ways of limiting diphtheria. The diphtheria cases which give a pure or nearly pure culture of the bacillus seem to be very fatal[1] unless efficient treatment is employed, so far as one may infer from 47 cases in which the mortality was 64%. A local treatment, which I shall describe later, is, I think, especially effi-

[1] It is important to realize that if the pulse in diphtheria suddenly changes during the acute stage of the disease and takes on the character indicative of a low blood-pressure, the prognosis is very grave, no matter how well the patient may appear to be in all other ways.

cient in cases where the Klebs-Lœffler bacillus only is found; nine cases giving a pure or almost pure culture of the diphtheria bacillus in which this treatment was used early all recovered.

As regards cases with false membrane in which the diphtheria bacilli are not found. In 50 cases of what may be called pseudo-diphtheria, occurring in different years, there was a mortality of 28%. As these cases were designated as pseudo-diphtheria on account of the absence from the bacterial test of the Klebs-Lœffler bacillus, we shall do well to look into this high mortality of 28%, or 14 out of 50 cases, a little more closely. These 14 cases, in about an equal number of instances, contained either streptococci or staphylococci, or both together.

10 were laryngeal, with 7 intubations,
1 had typhoid fever,
1 scarlet fever,
1 septic, with nasal discharge,
1 had nasal discharge.

14

Both the cases with nasal discharge were two years old, and in wretched condition when they entered the hospital.

Excluding the case of typhoid fever, there are two features which most of these 14 cases had in common; that is, their laryngeal character and their extreme youth — the average age was three years. It seems to me that these conditions determined the fatal outcome in these cases rather than any special coccus, or, what is more probable if we take the ground that the laryngeal cases are usually diphtheria, the Klebs-Lœffler bacilli probably escaped detection in some of them. As autopsies were not obtained, this point could not be determined.

In studying diphtheria one is struck by the large number of cases in which the larynx is involved and the high mortality among such cases. As we continue to use the bacterial test and obtain more autopsies I think we shall find that the percentage of fatal cases of membranous throats where the bacillus of diphtheria is absent will be smaller. Large numbers of cases of so-called pseudo-diphtheria already reported

by other observers, in which the mortality is very low, point to this conclusion. If the bacterial test has been carefully made, no bacilli of diphtheria found, and the larynx and nose are not involved, the prognosis of cases of membranous throats, where no other disease is present, — such as typhoid fever, pneumonia, measles, or scarlet fever, — is usually good. Of such cases my experience thus far has justified me in taking a hopeful view. I recall a case seen in consultation where the physician in charge had given a wholly hopeless prognosis, but the bacterial examination and the general condition of the patient led me to take an opposite view, which proved to be the correct one.

The inferences which may be drawn from suspicious cases seem to be these: First, cases which come to the hospital looking like diphtheria usually are such; that is, perhaps about nine times out of ten. The milder cases of membranous throats are not sent to the hospital in such large numbers as the severer ones, and thus the proportion of cases of diphtheria is smaller in suspicious cases if seen outside of the hospital; this has been my experience in private practice. Second, cases which look like tonsillitis, for example, usually are such, although now and then among such cases the bacilli of diphtheria are found. In practice it is well at first to regard everything which resembles diphtheria as diphtheria, until careful bacterial tests have been made.

Before leaving the subject of diagnosis, allow me to remind you again of the importance of recognizing the mixed cases of diphtheria and scarlet fever; these are doubly dangerous, as they may give rise to either disease in other individuals, especially in a hospital for contagious diseases, and require special and careful isolation. In 1892, when I first saw these mixed cases, that is, found the Klebs-Lœffler bacillus *coincidently* with scarlet fever in certain patients, their presence was denied by most of those, at least in this country, who had given the question special study, but now their existence has, I think, been generally recognized.[1]

[1] In scarlet fever it seems to me desirable to prevent the dissemination of the epithelial scales, which are often shed abundantly, and to hasten the removal of this layer of epithelium in order to free the normal skin. For this purpose I have my patients rubbed all over, daily, avoiding the hair, with a mixture containing seven parts of a 10-volume solution of hydrogen dioxide with one part of glycerine; if the

Three cases of typhoid fever with membranous throats in which bacilli of diphtheria were found, occurred during my service; one of these has already been referred to in a former paper of mine ("American Journal of the Medical Sciences," November, 1893), and Dr. John L. Morse has described a second of these cases (Isabella N.) in the Boston City Hospital Report for 1894. In my above-mentioned paper I also referred to the coincidence of diphtheria and measles during my service.

It is not very rare to see patients with syphilis presenting appearances in the throat similar to those found in diphtheria (of course both diseases may occur together); if syphilitic patches occur with an acute tonsillitis or pharyngitis the similarity to diphtheria is more striking.

Let us now consider the local treatment of diphtheria, first from the laboratory, and, second, from the clinical side.

The micro-organisms, dangerous above all others in the throat, are the venomous bacilli of diphtheria, and an early attack upon them with a suitable application diminishes the amount of the poison and probably prevents the extension of the bacilli to less accessible parts.

A rational step to be taken in our endeavor to destroy the bacilli is to ascertain what germicides will find and kill them quickly, and also which of the germicides, sometimes used, will not do so. I say find and kill, as there are many applications which will kill the bacilli in the laboratory, but when used in the throat simply wash the surface of the false membrane; there is one, however, which is an exception to this, as it has the quality of promptly disintegrating certain portions of the false membrane. In the plan which I adopted in the study of the action of various germicides upon the Klebs-Lœffler bacillus and several of the organisms which sometimes accompany it, I assumed that the germicide to be efficient must act quickly, as the contact in the throat between the germicide and the organisms is limited to a few seconds. It was, therefore, necessary to ascertain what would kill these organisms in five or ten seconds.

patient finds this too sticky the proportion of glycerine may be reduced. A quarter of the body may be done at one time, and some hours later another quarter, and so on. As soon as any portion of the skin is normal the application may be there dispensed with.

Second, if an agent or agents could be found to kill the organisms in five or ten seconds, the next step would be to ascertain what harmful effect, if any, they could have upon man, and to select one or more of those which would be least injurious to him: many germicides, such as corrosive sublimate, carbolic acid, the cresols, hydrochloric, nitric, phosphoric, and sulphuric acids alone and in efficient strengths are either poisonous or irritating, or both.

The third step in the investigation would be to learn how to apply efficiently a substance quickly fatal to the organisms and harmless to man, provided such a substance could be found.

Tests with various germicides were made under my direction, or by me, in the Biological Laboratory of the Massachusetts Institute of Technology upon the bacillus of diphtheria, the staphylococcus pyogenes aureus and albus, the streptococcus pyogenes, and a diplococcus, and I wish to express here my appreciation of Prof. W. T. Sedgwick's kindness in permitting me to use his laboratory, and my obligations to his former and present assistants, Messrs. McLauthlin, Mathews, and Keith, for carrying out many of these tests.[1]

[1] The following outline taken from a former article will show the method pursued. In any of the ordinary methods for the determination of the germicidal action of any liquid disinfectant upon a given bacterial species, three distinct and successive processes may usually be recognized. These are: first, the mingling for a known period of a pure culture of the given species with a suitable amount of the germicide; second, the dilution, after the desired interval has elapsed, of this mixture to the extent necessary not only to check further bactericidal action, but also to avoid the possibility of any future inhibitory effect; and third, the planting of a portion of this dilution in a suitable culture medium, in order to permit the growth and consequent recognition of any bacteria which may have survived the action of the germicide.

As a source of the diphtheria bacillus, we used a pure culture that came originally from the laboratory of Dr. Welch, of Johns Hopkins University, or one from Dr. Prudden's laboratory in New York, or from my own cases. From these, plantings were made in bouillon, and to ensure greater accuracy, in most of the experiments old and fresh bouillon cultures were used side by side, or a mixture of the two was employed.

The following record of a single action experiment with its control will serve to illustrate this method.

Five c.c. of 50-volume dioxide solution (1/6 % acid) were placed in a 60 c.c. Erlenmeyer flask and 0.25 c.c. added of a bouillon culture of the diphtheria bacillus. (In later tests, the germicide was poured over the culture.) Ten seconds after, in order to stop the action of the dioxide, the mixture was quickly poured into a litre flask containing 600 c.c. of slightly alkaline normal salt solution. Thirty seconds after this, the whole having been well shaken, 0.2 c.c. were transferred to 75 c.c. of alkaline normal salt solution in another flask. At the end of three minutes (in the

All of the tests given in the following tables, besides many others, have been made in duplicate, and some have been repeated several times.

TABLE I. — Tests with various Germicides upon the Klebs-Lœffler Bacillus.

Action period, 10 seconds.

Germicide.	Did not kill all.	Killed all.
[1] Hydrochloric acid	3%	4%
[1] Nitric acid		2%
[1] Sulphuric acid	5%	10%
[1] Phosphoric acid	2%	
Boric acid	4%	
Solution of chlorinated soda (hypochlorite of sodium), containing of available chlorine	1–2500	1–1000
[2] Chlorine	1–25000	1–10000
Corrosive sublimate	1–10,000 (0.01%)	1–5000 (0.02%)
Chloroform (in water)	1–200	
Carbolic acid	[4] 5%	[4] 5%
Tricresol in 25% alcohol		3%
[3] Metacresol in water		½%
Paracresol in water		½%

later experiments at the end of thirty seconds), 1 c.c. of the last dilution was planted in 6 c.c. of the bouillon. At the end of four and a half minutes 1 c.c. of the dilution was added to 6 c.c. of melted glycerine agar at 40° C., and the mixture at once poured into a Petri dish.

In the control experiment, 0.25 c.c. of the bouillon culture of the bacillus were mixed with 600 c.c. of alkaline normal salt solution, *then* 5 c.c. of 50-volume dioxide (⅙ % acid) added, and all were shaken. Thirty seconds after the addition of the dioxide, 0.2 c.c of the mixture were transferred to 75 c.c. of alkaline normal salt solution in another flask. After four and a half minutes, a glycerine-agar plate was made as before.

After a few days' growth in the incubator, the agar plate from the control experiment showed 1,600 colonies of the diphtheria bacillus. At the same time, neither the agar plate, nor the bouillon tube, prepared from the action experiment, showed any growth.

Evidently the agar plate is of especial advantage when a part only of the bacteria are killed, *i.e.*, when the germicide is diluted just beyond its effective limit. The bouillon tube gives no precise means of distinguishing between the survival of a part and the survival of the whole of the bacteria.

[1] U.S. Pharmacopœia dilute acid contains 10 per cent. of acid.

[2] Chlorine water contains 0.4 per cent. of chlorine, which diluted with 99 parts of water, gives 1–25000. Chlorine water cannot be mixed with hydrogen-dioxide solutions.

[3] I have used a solution containing ½ % of metacresol and 25-volume acid hydrogen-dioxide in twenty-five cases of diphtheria; objection has been made to the taste by some patients, and it seems to me to be less satisfactory than a strong hydrogen-dioxide solution alone.

[4] Does not always kill.

TABLE II. — Tests with various Germicides upon the Staphylococcus Pyogenes Aureus.

Action period, 10 seconds.

Germicide.	Did not kill all.	Killed all.
Hydrochloric acid	2%	4%
Nitric acid	½%	2%
Solution of chlorinated soda (hypochlorite of sodium), containing of available chlorine	1–2500	1–1000
Corrosive sublimate	1–5000	
Carbolic acid	1%	2½%
Tricresol in water	1%	1%
Metacresol in water	¼%	½%
Paracresol in water	½%	½%
Orthocresol in water	½%	

Staphylococcus Pyogenes Albus.

Germicide.	Did not kill all.	Killed all.
Hydrochloric acid	2%	4%

TABLE III. — Tests with various Germicides upon the Streptococcus Pyogenes.

Action period, 10 seconds.

Germicide.	Did not kill all.	Killed all.
Hydrochloric acid	⅛%	½%
Nitric acid		½%
Solution of chlorinated soda (hypochlorite of sodium), containing of available chlorine	1–2500	1–1000
Corrosive sublimate	1–10,000	1–5000
Carbolic acid	1%	5%
Metacresol in water	¼%	½%
Paracresol in water	¼%	

From these tables it will be seen that with the exception perhaps of the solution of chlorinated soda (hypochlorite of sodium) and the cresols, there is no one germicide that is equally active against all of these organisms. In general it may be said that the streptococcus pyogenes is the easiest to kill, the staphylococcus aureus the most difficult. A few tests

made upon the staphylococcus pyogenes albus indicate that in this respect it is similar to the aureus. A weak solution of hydrochloric acid, ½%, will kill the streptococcus in ten seconds, but it takes a 4% solution to kill the Klebs-Lœffler bacillus in the same time. It takes about the same strength to kill the staphylococcus aureus and albus. The staphylococcus aureus is more resistent to corrosive sublimate than these other organisms; on the other hand, it is more susceptible to carbolic acid than they are.

It should be remembered that in the laboratory tests the micro-organisms are presented to the germicides in a liquid culture, whereas clinically they are found enveloped in a mass of pus or false membrane, or other matter. Hence the advantage of using the substance I am about to consider.

To discuss the action of all the germicides I have studied during my work on diphtheria would not be feasible here, but I will devote a few words to one of them; namely, hydrogen dioxide.

The general principle in antiseptics which my studies have brought out, and which I believe to be a new one, is the advantage of mixing the stronger germicides with hydrogen dioxide, a substance which has the special property of disintegrating some kinds of dead organic matter, such as pus, or certain portions of false membranes, thus rendering the bacilli more accessible, and so opening the way for the action of the stronger germicides. Pure hydrogen-dioxide solutions, or rather neutral solutions, are comparatively weak germicides, but we may put with them any compatible germicides, or use them alternately with an incompatible one, the choice depending upon the micro-organisms we desire to kill. For instance, the mineral acids which are found in all the 10-volume hydrogen dioxide solutions sold in the market increase the germicidal value of these solutions. A 20-volume or even a 40-volume neutral solution of hydrogen dioxide will not kill all the streptococci in 10 seconds, but a weak solution of hydrochloric or nitric acid will do so. Tests, therefore, of the germicidal value of the hydrogen-dioxide solutions should always take into account the kinds and amount of free acid present.

TABLE IV. — Tests with Hydrogen-Dioxide neutral and acid Solutions upon the Klebs-Lœffler Bacillus.

Action period, 10 *minutes*.

Did not kill all.

$2\frac{1}{2}$ volumes containing . . . . . 0.05% of acid.[1]

This is stronger in acid than a U.S. Pharmacopœia 10-volume hydrogen-dioxide solution diluted with three parts of water, a strength which is frequently used by physicians.

Action period, 10 seconds.			Action period, 5 seconds.		
Did not kill all.			Killed all.		
5	volumes containing	0.1% acid.	50	volumes containing	0.17% acid.
[2] 10	" "	0.2% "	100	" " about	0.3% "
$12\frac{1}{2}$	" "	0.25% "	150	" " "	0.45% "
[3] 25	" "	0.5% "	200	" " "	0.6% "
50	neutral or slightly alkaline.		250	" " "	0.9% "

TABLE V. — Tests with Hydrogen-Dioxide neutral and acid Solutions upon the Staphylococcus Pyogenes Aureus.

Action period, 10 seconds.			Action period, 5 seconds.		
Did not kill all.			Killed all.		
25	volumes containing	0.5% acid.	100	volumes containing	1% acid.
50	" "	1.1% "	110	" neutral.	
80	" "	0.26% "			

Staphylococcus pyogenes albus was also killed by a 100-volume hydrogen-dioxide solution containing 1% acid.

TABLE VI. — Tests with Hydrogen-Dioxide neutral and acid Solutions upon the Streptococcus Pyogenes.

Action period, 10 seconds.

Did not kill all.			Killed all.		
10	volumes containing	0.05% acid.	25	volumes containing	1.1% acid.[4]
10	" "	0.10% "	$12\frac{1}{2}$	" "	0.5% " [4]
20	" neutral or slightly alkaline.				
40	" " " "	"			

[1] The acid was estimated as HCl with a 1-100 normal NaOH solution and phenolphthalëin.

[2] The U.S. Pharmacopœia 10-volume solution contains not more than 0.036% of acid.

[3] Sometimes a 25-volume solution containing 1-6 per cent. acid will kill, and sometimes it will not kill.

[4] The tests given in Table III., made later than these, show that 0.5 per cent. of hydrochloric acid alone "killed all" in ten seconds.

Tests with hydrogen-dioxide acid solutions upon a diplococcus from the throat of a patient ill with diphtheria showed that a 25-volume solution containing 0.1% acid did not kill in ten seconds, but that a 50-volume solution containing 0.1% of acid did kill in ten seconds.

It is not assumed that these bacterial tests have the accuracy of chemical ones. I have found nothing, however, which vitiates the general character of the deductions drawn from these tests: First, that neutral hydrogen-dioxide solutions are comparatively weak germicides; second, that the mineral acids, especially nitric acid, are stronger germicides; third, that hydrogen dioxide and hydrochloric acid together are more efficient than either alone against the Klebs-Lœffler bacillus; and fourth, that the 50-volume (0.2% acid) hydrogen-dioxide solution is not a good germicide against the staphylococcus, — 100-volume hydrogen dioxide acid (1%) solutions are required to kill this organism in five seconds. I hope to discuss the more general applications of these tests in another paper.

I observed an interesting correspondence between the laboratory tests and clinical experience in several cases, and will recur to this point.

We now come to the clinical side of the local treatment of diphtheria, and the treatment to which I wish to direct your attention is that with strong hydrogen-dioxide acid solutions.[1] The mortality in this State from diphtheria is greatest during October, November, December, and January, and my last service at the Boston City Hospital included the three latter months, November and December, 1893, and January, 1894, but I have permission to consider here the cases that were admitted in October, 1893, also.

On the mild, as well as on the hopeless cases, a 10-volume solution of hydrogen dioxide was generally used, alone or in connection with a solution of corrosive sublimate (1–5000 or 1–2500) or a solution of chlorinated soda. On *all* of the severer cases which came in the early days of the disease,

[1] I have found these strong solutions useful in minor surgery; they should not be used in closed cavities, as too much oxygen might be set free and cause pressure.

As a dressing for sores or ulcers, one may use a 10-volume solution of hydrogen dioxide containing 0.1—0.2 per cent. of hydrochloric acid; when desirable, pepsin may be added to this solution. The dioxide does not interfere with the action of the pepsin.

and during my service, I endeavored to use a 50-volume hydrogen-dioxide acid solution at short intervals.

To illustrate the advantages of using the 50-volume solution, let me take four patients who entered the hospital about the same time:

Two cases of diphtheria treated with a 10-volume solution of hydrogen dioxide.

P. H., 21 years old, entered the hospital December 1st. Klebs-Lœffler bacilli found in the cultures. Thick false membrane on both tonsils and on the sides of the pharynx. Under treatment of 10-volume hydrogen-dioxide solution every four hours the membrane had disappeared on December 20th, but there were still bacilli of diphtheria present on December 20th and 23d. The membrane returned again on January 1st. On January 3d no bacilli were found.

H. N., 6 years old, entered the hospital on December 2d. Klebs-Lœffler bacilli found in the cultures. Small specks of false membrane on right tonsil and right palate. Ten-volume solution every three hours. Bacilli still present on December 25th; absent January 5th.

Two cases of diphtheria: the first one treated in part and the other wholly with a 50-volume solution of hydrogen dioxide.

S. M., 10 years old, entered the hospital on December 3d. Bacilli of diphtheria found in the cultures. This case seemed more severe than either of those above mentioned. False membrane on both tonsils, left palate, right anterior pillar, right and left wall of pharynx. At first a 10-volume solution was used, but as the membrane increased a 50-volume solution was substituted. Bacilli of diphtheria present December 10th; absent December 16th.

J. W., 6 years old, entered the hospital December 8th. Bacilli of diphtheria found in cultures. A more severe case than any of the above. Thick false membrane on both tonsils, and on both anterior pillars. The next day it covered the right, left, and posterior pharyngeal wall. The use of fifty-volume hydrogen-dioxide acid solution was then begun. Bacilli of diphtheria were present on December 11th, throat clear on December 16th, bacilli absent December 17th.

Of these four cases the last two to enter the hospital pre-

sented more severe local symptoms than the first two, the last one especially. The patients who entered the hospital December 1st and 2d, respectively, had a 10-volume hydrogen-dioxide solution applied, and the bacilli were absent January 3d and 5th respectively. Average duration of bacilli, 34 days. The patients who entered the hospital December 3d and 8th, respectively, had 50-volume hydrogen-dioxide acid solution and the bacilli were absent December 16th and 17th respectively. Average duration of bacilli 11 days.

The three following cases further illustrate this point. The patients were adults of about the same age.

Case No. I. — Membrane on tonsils and posterior pharynx. Cultures were made from the throat about every four days and bacilli of diphtheria found. On the 53d[1] day of the disease bacilli of diphtheria still found in the cultures; on the 56th day none were found. The treatment in this case was 10-volume hydrogen-dioxide solution every hour, corrosive sublimate 1–2500 on a swab every two hours. Finally some of the strong hydrogen-dioxide solution was used; after this had been applied for about three days the bacilli disappeared.

One case of diphtheria in which a *partial* use of the strong dioxide acid solutions was made.

Case No. II. entered the hospital more than two weeks later than No. I. Membrane on both tonsils. Klebs-Lœffler bacilli found in the cultures. As there was no strong dioxide solution at hand a 10-volume dioxide solution was used every two hours, followed by 1–2000 corrosive sublimate with 1% citric acid on swab. During the treatment the membrane spread and covered besides both tonsils, the soft palate on both sides, anterior and posterior pillars, and the left and posterior wall of the pharynx. As soon as the strong dioxide arrived, the above treatment was stopped and a 50-volume hydrogen-dioxide solution was applied every two hours by day and every three hours by night for three days; then the intervals between the treatments were in-

[1] The duration of the bacilli in this case is above the average duration of the bacilli in the 23 cases alluded to later, in which the treatment was a 10-volume solution of hydrogen dioxide alone, or in connection with corrosive sublimate or chlorinated soda; but in two other of these cases the bacilli were present in the cultures three weeks longer than in this case.

creased. After the culture made for diagnosis, no subsequent culture, unfortunately, was made until the 26th day of the disease. No bacilli were then found or afterwards. This patient was discharged from the hospital two weeks earlier than No. I., and entered, as above stated, more than two weeks later.

One case of diphtheria, in which a *complete* use of the strong hydrogen-dioxide acid solutions was made.

Case No. III. — This case entered the hospital a month after No. I. Klebs-Lœffler bacilli found in the cultures. The membrane was everywhere on tonsils, palate, uvula, pharynx, both sides and back, and in nose, — a case which justified the old-fashioned term of putrid sore throat. A 50-volume hydrogen-dioxide solution was applied in spray every three hours during the day and twice during the night for five days; a 25-volume solution was then substituted, which was applied three times during the day. No bacilli were present in a culture made on the 8th day of the disease nor at any subsequent examination. The throat of No. III. was free from bacilli two weeks earlier than No. I., although the patient entered the hospital a month later than No. I.

An estimate of the advantage in using the 50-volume solution, in point of the duration of the bacilli, is shown better by comparing the average time during which the bacilli remained in the throats of the milder cases which were treated with a 10-volume solution of hydrogen dioxide alone, or in connection with a solution of chlorinated soda or corrosive sublimate, with the average time during which the bacilli remained in the throats of patients, more severely ill, but who on the other hand were treated with 50-volume hydrogen-dioxide solutions. Most of both sets of cases occurred during about the same months.

In the first class of cases, 23 in number, the bacilli were found on the average to be present in the cultures on the 27th day of the disease and absent on the 36th day of the disease. No cultures were made between the two periods from which these averages were made up. The average age of these cases was 17 years.[1] In the second set of cases

[1] Since the above was written I have used 0.5 per cent. salt solutions in 11 cases. In these the bacilli were found to be present on the average on the 26.3 day of the dis-

(severer ones), namely, those treated with strong hydrogen-dioxide acid solutions, 26 in number,[1] the bacilli were found on the average to be present in the cultures on the 12th day of the disease and absent on the 18.8 day of the disease. No cultures were made between the two periods from which these averages were made up. The average age of these cases was 19.4 years.

It is hardly necessary to point out that the strong dioxide would probably have driven the bacilli from the throats of the milder cases sooner than it did from the severer ones.

In two-thirds of the 26[1] cases treated with strong hydrogen-dioxide acid solutions, the *bacilli* disappeared *before* the *membrane;* in the remaining third this point was not determined. The patients were not discharged from the hospital until both bacilli and membrane had disappeared.

The capacity of a hospital for the treatment of diphtheria would be increased by using the local treatment with strong hydrogen-dioxide solutions rather than those ordinarily employed, as the patients would remain in a diphtheria ward a shorter time.

The *diminution* of the bacilli in the throat under different treatments may be studied by examining a cover-glass preparation under the microscope made from swabs taken directly from the throat. This was done in a few cases only. After a patient had had, for example, a **10**-volume hydrogen-dioxide solution for ten days, bacilli of diphtheria and other organisms were readily found. In cases treated with **50**-volume hydrogen-dioxide solution examined in the same way from two to seven days after beginning treatment, very few organisms of any kind were found; bacilli were, however, found in cultures in these cases for a longer period than in the cover-slips; these examinations show the marked and rapid diminution in the number of bacilli under the treatment by a 50-volume hydrogen-dioxide solution.

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ease and absent on the average on the 35.4 day of the disease. No cultures were made between the two periods from which these averages were made up. One of these 11 cases had what may be described as a raw-beef throat — a condition which lasted nearly as long as the bacilli remained in the throat, which was six weeks.

[1] These cases are 26 of the 30 cases taken up on page 31; of the four remaining cases, two died and two had no bacterial test applied before discharge; therefore they could not be taken into consideration here.

The following case is one of those which illustrates how the 50-volume hydrogen-dioxide acid solution may kill the diphtheria bacilli in a wound:

A patient ill with diphtheria had a contused wound, which was incised and the pus let out. A swab was taken from the wound and the culture made from it was an almost pure culture of diphtheria bacilli. The flaps of thick dead skin were trimmed off, leaving an open wound about an inch in diameter, covered with pus. The wound was cleansed with 50-volume hydrogen-dioxide acid solution on a mop and covered with gauze moistened with a 10-volume solution. Four hours later the gauze was removed and a second swab taken; no diphtheria bacilli were found, only staphylococci. That the diphtheria bacilli were killed by the 50-volume hydrogen-dioxide solution and the staphylococci were not, is in accord with the laboratory tests.

Since this paper was read, by the kindness of Dr. H. W. Cushing I have used in certain cases in his service (surgical) the strong hydrogen-dioxide solutions—usually 25, sometimes 50 volumes — freely upon large granulating surfaces of burns and ulcers; when they were so situated that the strong dioxide solution could be poured into a cuplike depression of the granulating surface, this was done, and the dioxide allowed to remain some minutes; it was then washed out and more strong dioxide solution poured in; this was repeated several times. The granulating surfaces were cleansed in this way daily for weeks without causing the patient any discomfort, and were dressed with a dilute solution of hydrochloric acid $\frac{1}{10}$ or $\frac{1}{5}$ %. All healed well.

Let us now consider the METHOD of applying the strong hydrogen-dioxide solutions.[1]

The Strength of the Applications. — This should ordinarily be from 25 to 50 volumes, according to circumstances,

[1] How not to use the solutions of hydrogen dioxide in diphtheria is very well illustrated, I think, by some cases that were brought to my attention since this paper was read. A solution of hydrogen dioxide in ether, known as pyrozone, was used to make the hydrogen-dioxide solution. The manufacturers claim that these solutions are nearly neutral; in other words, an important germicidal element, the acid, is left out; the special property of the hydrogen dioxide itself being the disintegrating power. Second, the air-pressure used in the atomizers was too great. The air was pumped up to a pressure of 80 pounds, then allowed to run down to 30 pounds and again pumped up to 80 pounds. The pressure should not be more than 6 or 8 pounds.

containing about $\frac{1}{10}$ to $\frac{1}{5}\%$ of acid. A stronger solution must be used in the throat than in the laboratory on account of the dilution by the saliva. If there is much membrane, and especially if the case is septic and in the early days of the disease, the full 50-volume strength is needed. It is well to make the application with a mop so far as may be, and the spray should be used gently. One should not omit to treat the nose and keep it clear, but a separate atomizer should be used for this purpose. In spraying the nose, only very weak solutions **1** or **2** to **5** volumes should be used. The spray should be directed horizontally into the nostril and not upwards. In the posterior nares the strong solutions may be employed by means of an up-spray, or by a spray directed forwards from the posterior nares; the formation of much foam after the application of the solution to the latter parts indicates the presence of membrane. It is very important to know the acidity and strength of the solutions of hydrogen-dioxide, and the value of the treatment cannot be properly estimated unless analyses of the solutions are made. The different makes vary in the amount of acid and hydrogen dioxide they contain. The 10-volume solutions purchased for the hospital, from which the strong solutions were made, have been excellent. The most efficient mode of application is by means of a gentle spray under an air-pressure of 6 to 8 pounds, just sufficient to carry the solution to the desired spot; this avoids dilution better than when the applications are made on a mop, although sometimes a mop answers well, and it is easier for the patient.

The discomfort of all local applications is usually greatest during the early days of the disease, when the throat is most sensitive and most congested, but at this time treatment is most serviceable. Later in the disease when the false membrane is clearing off, and if a sensitive, raw, congested surface is exposed, especial care should be taken to avoid these sensitive areas or use the dioxide solution on a mop. One should be careful not to cause bleeding. The applications, especially in spray, should be made most gently. If the mop is used, the solution causes much less, sometimes no discomfort. The amount

of discomfort is, in most instances, an indication of the want of skill in making the applications. While the smart of the strong dioxide is usually greater at the time than that of some of the other applications which are made to the throat, it is only momentary; patients invariably prefer the strong hydrogen-dioxide solutions to chlorinated-soda solutions of efficient strength. I recall some patients who spoke of the pain the strong dioxide solution caused, and I therefore tried, without their knowledge, spraying their throats with water, and often they did not distinguish between the two. It is the friction of the spray which I think chiefly causes the discomfort, and some throats are evidently more sensitive than others, and some patients more nervous. The majority of patients do not mind the strong dioxide solutions, but many ask to have them applied oftener on account of the relief which follows their use. In an adult where the nose was closed with false membrane and the tonsils so much swollen and covered with false membrane as to cause cyanosis, the throat was cleared in a few moments by the use of strong hydrogen-dioxide solutions, and the breathing was thus made perfectly easy, although a few moments before tracheotomy had been considered.

The Number of Applications.— The strong hydrogen-dioxide solutions should be applied often during the first few days; by often I mean once every two hours by day, and twice during the night for the first night or two in severe cases. It will be found, after a few days, in some cases after two or three, that the diphtheria bacilli are much diminished in number or have even disappeared from the throat, although much false membrane may remain. Their disappearance can only be determined by cultures; false membrane in the throat is not a proof of the presence of the bacilli of diphtheria; it may be due to other organisms. No matter how much membrane there may be in the throat, we may be much relieved when the bacilli have disappeared from the cultures. When this has taken place local treatment should be omitted, except to give comfort to the patient by clearing the throat.

I am inclined to think that the persistence of the Klebs-Lœffler bacilli for more than a week or ten days, under the

strong hydrogen-dioxide treatment, is due usually to their being concealed in the folds of mucous membrane or in pockets which have been made by the inflammatory process, especially in the tonsils, and that the attention should be directed to the application of the solution to these hiding-places carefully and thoroughly.

I observed that in some cases of diphtheria the false membrane persisted under the use of the 50-volume hydrogen-dioxide acid solution for some time after the Klebs-Loeffler bacilli had ceased to be present in the cultures; staphylococci were, however, present. This led me to study the action of the 50-volume solution upon the staphylococcus and to find that it would not kill this organism readily in the beaker. It will be seen in Table V. that a 100-volume hydrogen-dioxide acid (1%) solution is required to kill the staphylococcus in five seconds. Further, I found that chlorinated soda would kill it in the beaker quickly (10 seconds), and that clinically the false membrane that had persisted under the use of a 50-volume solution yielded under the alternate use of the 50-volume solution and a solution of chlorinated soda containing from 1–1000 to 1–500 available chlorine. I have tried this in a few cases only.

The local treatment of membranous throats may, therefore, be best carried on with the microscope and culture tube in one hand and the remedies in the other. As soon as the bacilli of diphtheria have disappeared the strong dioxide should be omitted, except as a cleansing, deodorizing,[1] or disintegrating agent. If we find that the organisms present are chiefly staphylococci we may substitute chlorinated soda for the strong dioxide, or use them alternately, — they should never be mixed nor used too near together, — or we may use hydrogen dioxide and carbolic acid mixed together. The 50-volume dioxide does not kill the staphylococcus readily in the beaker; it will not, therefore, in the throat.

It follows from what has been just said that in treating

[1] A 5 per cent. solution of permanganate of potassium is frequently used as a deodorizer; a saturated solution contains about 6 per cent. of this substance; this would correspond as a source of oxygen to about a 6-volume solution of hydrogen dioxide.

pseudo-diphtheria where staphylococci are present we may use hydrogen-dioxide solution as a disintegrating agent, and chlorinated soda, or carbolic acid, as germicides.

In passing let me add a word of caution as to the use of alkalies in diphtheria. Many practitioners prescribe lime-water in diphtheria, and many are in the habit of prescribing some simple local application containing bicarbonate of soda, such as Dobell's solution, in a case of sore throat. Such alkalies seem to me disadvantageous in diphtheria, as they favor the growth of the bacilli and the formation of their poison; we should, therefore, be careful to avoid them in this disease or where there is a suspicion of the disease. It seems better to have the diseased parts bathed in acid, not alkaline, solutions.

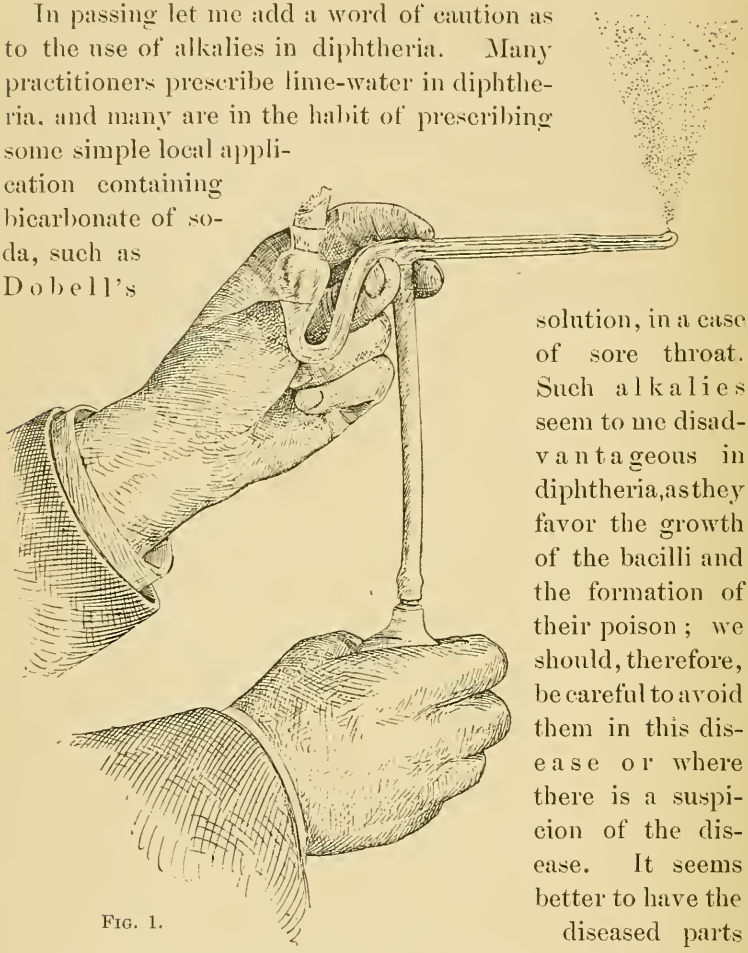

Fig. 1.

From the thumb to the point the tube is 5 inches long. The up-spray is shown in this figure. The top-shaped bulb should not be filled more than one-half full. The mouth of the bulb may be loosely closed with a small roll of gauze (not absorbent cotton) to prevent spattering.

The instruments which I have designed and found efficient for applying the hydrogen-dioxide solutions are, first, three glass atomizers which can be readily sterilized by boiling. One of these has a down-spray, the second a

return-spray, and the third an up-spray. The latter (see Fig. 1) may be used to direct the spray to the side and upper portion of the pharynx or to either tonsil without taking it out of the mouth, simply by turning it in the hand. A spray is best applied to the surface of the tonsils if directed at right-angles to it. This can easily be done with this atomizer, which I find indispensable. The rubber bulbs of these atomizers have two valves. I also use an atomizer with a straight spray.

The atomizer for spraying the posterior nares from behind forwards (return-spray) is made by bending the atomizer, shown in Fig. 1, through ninety degrees one-half an inch from its tip.

The mop which I use for swabbing the throat consists of a small glass rod on which some absorbent cotton has been twisted. The rod is about eight inches long and one-eighth of an inch in diameter, and is bent at an angle of about 45 degrees half an inch from the end, in order that the sides of the pharnyx may be more easily reached. The end of the rod is rounded and thickened a little by heating in a flame, as by this device the cotton is held more firmly.

To make the applications with success we should not only have good instruments, but also the coöperation of the patient; it is a mistake to use force — I do not mean that young children should not be held in a position to let a good light fall into the throat, but the mouth should not be forced or pried open. One can nearly always gain the coöperation of children who are four or more years old (I have used this treatment on children two years old who took it satisfactorily). If one begins tactfully it is surprising to see how soon young children accept the habit of having the throat treated. It is not necessary to have the head lifted from the pillow. In order to accustom the patient to open the mouth properly I sometimes go through the manipulations of treatment once or twice with water instead of the remedy. It is very important to have the part which is to be treated well lighted, so that one may *see exactly* what is *required* and also what is *done*. If the physician does not apply the treatment himself, he should see it done whenever he can. At night a very small kerosene lamp with a reflector

which also shades the eyes of the physician, a necessary condition to success, is good. I sometimes use a small electric-light with a portable battery. It is not easy to apply local treatment properly in the throat, and the best results can only be obtained when great care is taken.

In looking over what I have written it seems to me necessary to add some details essential to success in carrying out the treatment with *strong* hydrogen-dioxide acid solutions, as there seems to be so much misconception about them as well as about the ordinary 10-volume solutions. Neutral solutions do not keep well, and alkaline ones decompose readily. The hydrogen-dioxide solutions which are sold in the market all contain some acid which increases their germicidal value and assists in preserving them.

The strong solutions of hydrogen dioxide may be made by evaporating the 10-volume solution (U.S. Pharmacopœia, 1890) in a porcelain dish over a stove or Bunsen burner; in this way one may, if one desires it, obtain a liquid containing 90 odd per cent. of hydrogen dioxide. A 50-volume solution (about 16%) of hydrogen dioxide may be readily obtained by evaporating a good 10-volume solution to $\frac{1}{6}$ of its weight; a 25-volume solution, by evaporating a good 10-volume solution to $\frac{1}{3}$ of its weight.[1]

[1] If there were no loss of hydrogen dioxide during evaporation, this process would yield a 60-volume solution, but about 20 per cent. of the dioxide is lost; therefore, in making a 50-volume solution it is necessary to evaporate a 10-volume to one-sixth instead of one-fifth of its weight. In making solutions of greater strength, the loss would be rather more; in making those of weaker strength, say 25 volumes, the loss would be less than 20 per cent. There is no economy in buying a solution that has not been well made, as it does not keep well and loses more on evaporation than a good one. The time required to evaporate over a gas stove 190 grammes of a 10-volume solution (U.S. Pharmacopœia, 1890), to 32 grammes (an ounce), of 50-volume solution, is half an hour. One may carry on the evaporation at a gentle boil at the rate of about 6 grammes (one drachm and one-half) per minute.

For commercial purposes the strong solution can doubtless be made with a smaller amount of loss. I simply suggest how in the emergency of diphtheria one may quickly obtain a strong solution. Usually I make some ounces at one time.

In the solution of the Oakland Chemical Company the free acid seems to be chiefly hydrochloric, and this is the acid which I now prefer. It is a better germicide than sulphuric acid, although not so good as nitric. Hydrogen-dioxide solutions keep better in cool than in warm places; they decompose more rapidly in sunlight.

I have lately made twenty-six experiments, extending over periods of from four to twenty-six weeks, upon the keeping qualities of hydrogen-dioxide solutions of strengths varying from 10 to over 100 volumes, and these tests show that the strong, 25 and 50 volumes, hydrogen-dioxide solutions of good make are sufficiently stable to be perfectly practicable.

Hydrogen dioxide is a bleaching agent and should not be brought into contact with

In making the strong solutions it is not necessary to add any acid, as but little of that present in the solution is lost, and 10-volume solutions containing 0.030% of acid when evaporated to $\frac{1}{6}$ of their weight contain about 0.18% of acid, and this is as much as is required for use in diphtheria.

Let me remind you in passing that the gastric juice contains 0.20% of hydrochloric acid. As this amount does not injure the lining membrane of the stomach, it could hardly injure the throat when applied for a short period only.

Bromide of potassium is of much service to adults during the first few nights, as they are very anxious when they find they have so severe a disease as diphtheria; children do not need it, as they go to sleep directly after treatment, or if asleep are frequently not fully awakened by treatment.

In many cases of retraction much relief follows the inhalation of steam or calomel fumigations.

There should be abundant and dry air in the patient's room and abundant and direct sunlight. Sunlight is especially advantageous in a diphtheria ward on account of its excellent germicidal properties, and every room in a hospital for such patients should have this. The simplest construction will permit these conditions to be fulfilled. The building should run north and south so that every bed may have sunshine part of the day. The utmost attention should be paid to the nourishment of the patient; if too little food is taken by the mouth it should be supplemented by feeding in other ways, as by the rectum or by means of a soft catheter and a funnel. The catheter may be inserted gently through the nose into the œsophagus and liquid food may be poured into the funnel.

Good food, absolute rest, protection from draught, or from a temperature that might give the patient a cold, are to be insisted upon. In some cases stimulation may be desirable. Strychnine, digitalis, and iron should be used when required.

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the hair or colored fabrics. One should avoid getting it on the skin, as it may, after a few minutes, whiten the skin and cause a pricking sensation. It is probable that it attacks the dead outer layer of the epidermis, and hence the white appearance; a minute portion of the solution may get into some small cavities of the skin, and the oxygen gas be there liberated, thus causing pressure on the endings of the sensitive nerves.

The mortality from diphtheria differs in different cities (in recent years it has been very much higher in Boston than in some other cities) and in different institutions, and may differ in the same institution during the same period of time. These variations, I believe, *usually* depend not so much upon the special medical treatment employed as upon the state in which patients are admitted and the conditions which surround them after admission; by this I mean the ventilation of the ward, the amount of sunlight, the care taken in regard to the cleanliness of the instruments, exposure to other diseases, etc. The milder cases recover if no harmful treatment is employed.

At the Boston City Hospital there are two buildings for the scarlet-fever patients; one belonging to the hospital which is used for acute cases of scarlet fever, and the other, a wooden building, placed at the disposal of the hospital less than a year ago, which is used for the convalescent cases of scarlet fever. Diphtheria has developed in both buildings, but in the first, the cases have usually been mild in character; in the second, the building for convalescents, they have been severe, among the most severe and fatal we have had at the hospital. The two buildings are erected on land that is only a few feet above high-water mark, but in the building that does not belong to the hospital the damp ground is not covered with cement as is the case in the other building.

I have used the strong hydrogen-dioxide acid solutions in over 100 cases, which varied in age from 2 to 68 years; some were cases of pseudo-diphtheria, and others were hopeless cases of diphtheria in which it was used to give comfort to the patient by getting out membrane from the throat, so that swallowing was made easier, or to keep down the septic odor; again, others were cases that came under treatment early in the disease.

These solutions fulfil the conditions sought: they will kill the bacilli of diphtheria in the laboratory in five seconds, and are not poisonous (they are readily decomposed into oxygen and water); they prevent the septic odor, and take off certain portions of the false membrane better than anything else I have employed; under their use the congestion and glandular tenderness and enlargement is

more quickly diminished, and the bacilli remain for a shorter time in the throat. Not only is the course of the disease shortened, but also the convalescence is conspicuously rapid compared with the cases under my care before I used this treatment. The patient is thus for a shorter time a menace to those around him, and the danger of the spread of the disease is much lessened. There is conspicuously less tendency to cardiac weakness.

In studying the effect of any local treatment upon the mortality it seems to me that we should consider chiefly the cases in which the treatment has not been delayed too long, say not beyond three days.

I will, therefore, discuss here two series of cases of membranous throats, occurring in different years, that were treated with strong hydrogen-dioxide solutions and comply with this condition. There were 18 in the first series, and 30 in the second series; 48 in all. During the first period there were, in all, 23 cases of membranous throats, the diagnosis of which was not based on cultures, that entered the hospital within the first three days of the disease, so far as known. Five of these do not concern us, as they were not treated with strong solutions of hydrogen dioxide, but being operative cases, were transferred directly from the medical to the surgical side of the hospital, were there intubed, and died two days after entrance. The remaining 18 make up the first series of cases treated with strong hydrogen-dioxide acid solutions within the first three days of the disease. One of these 18 cases died; this was a laryngeal case at entrance.

During the second period there were, in all, 40 cases of diphtheria, the diagnosis of which was based on cultures, that came under my care within the first three days of the disease so far as known. Ten of these cases do not concern us, for the following reasons: Seven were mild or very mild cases that recovered and were not treated with strong hydrogen-dioxide acid solutions. The eighth was a child who entered the hospital on the *second* day of the disease, but on account of extreme homesickness did not have any treatment until the *fourth* day of the disease. She was then treated with the strong hydrogen-dioxide acid solutions and recovered. The

ninth and tenth cases were not treated with the strong hydrogen-dioxide solutions until after three days, as there was none in the ward at the time, or it was deficient in strength. These two cases died. The remaining 30 cases of diphtheria, making up the second series of cases under discussion, were treated with strong hydrogen-dioxide acid solutions within the first three days of the disease, so far as known. Four-fifths of these 30 cases entered the hospital during the months in which the mortality from diphtheria in Massachusetts is greatest. Of these thirty cases two died. One was a woman, 24 years of age, who was vaccinated a few days before the attack of diphtheria began, and the solution used in this case was found to have been much below 50 volumes in strength. (In private practice I always test the solutions before using them.) The patient some years before had lost a brother and sister with diphtheria. The other was a child four years of age, who took the treatment well for one day, but became delirious the next day, so that further treatment was impracticable, and died the following day.

It gives me pleasure to testify here to the careful manner in which the house officers and nurses carried out my instructions in regard to these patients.

I have used the strong hydrogen-dioxide acid solutions at the Boston City Hospital during the years 1892, '93, and January, '94, six months in all, as well as in private practice, and have used them with increasing satisfaction.

In closing this paper I wish to emphasize the importance of the early diagnosis of diphtheria, and, if the treatment be local, of prompt, gentle, and thorough treatment during the early days of the disease with strong hydrogen-dioxide solutions of known strength, containing a proper amount of acid, applied with efficient instruments.

APPENDIX.

Since this paper was written I have used anti-toxin in a few cases ("Boston Medical and Surgical Journal," December 20, 1894) with excellent results, and initiated its use at the Boston City Hospital, where its administration in this community was begun. Under this treatment the bacilli often disappear from the throat early. In three of the cases an eruption appeared several days after the anti-toxin was injected; in three there was pain in the bones; one of these had periostitis of the ulnar and an extremely painful tenosynovitis about the left hand and wrist; two had swelling of the joints.

The syringe which I have designed and use to inject the anti-toxin (see Fig. 2) is a modification of the one I published in 1892; it is made of glass and packed with asbestos, and is joined to the hypodermic needle by a short piece of rubber tubing. By this device the anti-toxin is injected with greater ease into a struggling child, and the danger of breaking off the needle under the skin — an accident which may happen when the needle is rigidly fixed to the syringe — is avoided. This instrument may be sterilized by boiling in water.[1]

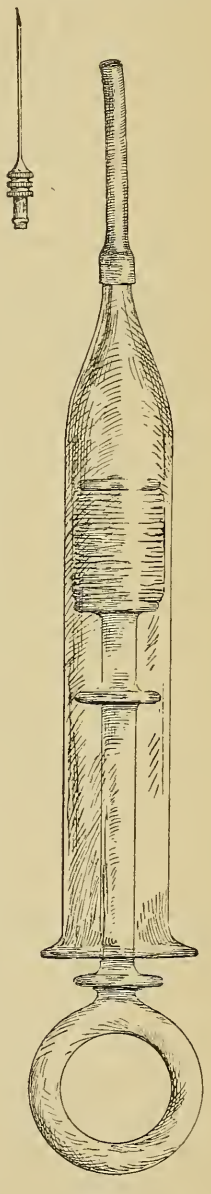

Fig. 2.

In Europe, local treatment is recommended to be used with anti-toxin. In some of my patients I have used anti-toxin alone, and in others I have combined with it local treatment with a 25-volume acid solution of hydrogen dioxide in the throat (made from the U.S. Pharmacopœia 10-volume solution); and in nasal diphtheria I have used in the nose a dilute (2-volume) solution. These solutions may

[1] To fill the syringe remove the needle, insert the rubber tube into the bottle of

be used every three hours in spray. The loosening and softening action of the anti-toxin, together with the disintegrating action of the hydrogen-dioxide solution, remove the membrane rapidly, and the hydrogen-dioxide solution lessens, if it does not obviate, the danger of pieces of false membrane getting into the trachea from the pharynx.

In treating diphtheria I now usually inject anti-toxin, and apply a spray of a 25-volume acid solution of hydrogen dioxide locally in the throat, and a 2-volume solution in the nose once in three or four hours.

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serum and draw in the required amount; then hold the syringe vertically, with the rubber tube up, draw in air enough to fill the conical end of the syringe and put in the needle; next hold the syringe horizontally and fill the needle with serum. Insert the needle well under the skin, and inject the serum, holding the handle up; the bubble of air near the piston allows all the serum in the syringe to be injected. This syringe is made by Codman & Shurtleff, Boston, and is inexpensive.

III.

HEAT PROSTRATION. AN ANALYSIS OF ONE HUNDRED CASES OCCURRING IN THIRTEEN CONSECUTIVE YEARS.

By Charles F. Withington, M.D.

The dangers arising from heat prostration affect most particularly that portion of the community which is obliged to work at all times, irrespective of weather and in exposed situations, — in other words, exactly the class of persons for whom a hospital is especially designed. Hence, probably, a larger proportion of the cases of sunstroke and heatstroke which occur in the city are brought to the City Hospital than of any other accident to which the community is liable. It has seemed, therefore, useful to collect some facts from the hospital records bearing upon insolation. The records of the various medical services have been examined from the year 1882 to 1894 inclusive, and all the cases of heat prostration tabulated. These numbered a little over a hundred, but after rejecting a very few in which the evidence was not perfectly satisfactory as to the nature of the illness, or where the records were incomplete, there remained exactly one hundred well-marked cases of insolation, upon which the following report is based.

As was, of course, to be expected, the male sex was affected in a vastly preponderating degree. There were 95 males and 5 females. The oldest patient was 70, and the youngest 19, the average age being about 30, and representing that of the most laborious portion of the community.

The time of day at which the attack occurred was deemed of sufficient interest to be noted, and it appears that most of the cases happened in the afternoon. Fifty-two are recorded as occurring after noon, as against 15 in the forenoon, 3 at noon, and 1 in the night. Two to three P.M. represents the most frequent hour of the day, as would be natural,

after several hours of heat have overcome the resisting capacity of the organism.

The great variation in the number of cases of insolation in consecutive years is illustrated by the following table:

CASES BY YEARS.

1882 . . . 4	1887 . . . 19	1892 . . . 16
1883 . . . 8	1888 . . . 9	1893 . . . 9
1884 . . . 8	1889 . . . 1	1894 . . . 14
1885 . . . 3	1890 . . . 2	——
1886 . . . 2	1891 . . . 5	100

The distribution of the cases as regards the date of their occurrence is shown by the following table, in which are incorporated also the meteorological facts of maximum and minimum thermometer, and the mean humidity. These data are furnished through the kindness of Mr. J. W. Smith, Local Forecast Officer at Boston, from the official records of the U.S. Weather Bureau.

Date.	No. of Cases.	Max. temp.	Min. temp.	Mean rel. hum.	Date.	No. of Cases.	Max. temp.	Min. temp.	Mean rel. hum.
July 9, 1882 . . .	1	87	65	69	June 24, 1888 . . .	1	78	57	88
" 27, 1882 . . .	1	98	68	56	July 3, 1888 . . .	1	74	56	56
" 29, 1882 . . .	1	92	68	59	Aug. 2, 1888 . . .	1	87	61	58
Aug. 9, 1882 . . .	1	89	68	67	" 18, 1888 . . .	1	84	68	67
June 6, 1883 . . .	1	91	65	82	July 30, 1889 . . .	1	85	72	90
July 4, 1883 . . .	1	96	64	68	" 31, 1890 . .	2	95	76	74
" 6, 1883 . . .	1	96	69	67	June 15, 1891 . . .	1	95	67	52
" 7, 1883 . . .	1	96	74	63	" 16, 1891 . . .	2	96	73	52
" 12, 1883 . . .	1	87	59	71	Aug. 12, 1891 . . .	1	90	72	80
" 16, 1883 . . .	1	78	62	87	" 25, 1891 . . .	1	88	74	80
Aug. 24, 1883 . . .	1	82	68	53	June 13, 1892 . . .	2	94	72	59
June 6, 1884 . . .	1	89	63	65	" 14, 1892 . . .	2	96	74	66
Aug. 18, 1884 . . .	1	94	68	82	July 13, 1892 . . .	2	91	70	73
Sept. 5, 1884 . . .	4	90	71	61	" 14, 1892 . . .	1	87	72	70
" 6, 1884 . . .	1	88	66	61	" 25, 1892 . . .	2	94	73	69
" 10, 1884 . . .	1	94	71	75	" 26, 1892 . . .	3	96	74	62
July 22, 1885 . . .	1	86	67	60	" 27, 1892 . . .	2	93	72	47
" 25, 1885 . . .	1	91	73	76	Aug. 9, 1892 . . .	1	89	71	78
Aug. 13, 1885 . . .	1	86	73	80	" 10, 1892 . . .	1	94	76	66
July 8, 1886 . . .	1	81	70	62	June 5, 1893 . . .	1	91	65	72
Aug. 10, 1886 . . .	1	86	64	62	" 6, 1893 . . .	5	90	73	78
May 10, 1887 . . .	1	86	48	51	" 14, 1893 . . .	1	70	58	96
" 24, 1887 . . .	1	77	64	76	" 15, 1893 . . .	1	82	66	87
July 2, 1887 . . .	5	95	72	63	Aug. 25, 1893 . . .	1	93	68	71
" 3, 1887 . . .	1	88	71	76	June 16, 1894 . . .	3	92	66	68
" 13, 1887 . . .	4	92	66	63	" 17, 1894 . . .	2	95	72	55
" 25, 1887 . . .	1	87	74	75	" 22, 1894 . . .	1	88	66	70
" 26, 1887 . . .	1	84	68	86	" 23, 1894 . . .	2	94	72	58
" 27, 1887 . . .	1	78	62	91	" 26, 1894 . . .	1	88	66	84
Aug. 1, 1887 . . .	2	80	70	74	" 28, 1894 . . .	1	87	69	71
" 9, 1887 . . .	2	77	58	65	July 2, 1894 . . .	1	86	72	74
June 7, 1888 . . .	1	81	56	73	" 20, 1894 . . .	3	95	72	65
" 11, 1888 . . .	1	81	61	66	" 21, 1894 . . .	1	87	65	74
" 22, 1888 . . .	1	88	55	79	Aug. 8, 1894 . . .	1	90	67	67
" 23, 1888 . . .	2	96	69	66					

From the foregoing table it is seen that the earliest date of any sunstroke was May 10, and the latest August 25.

The following extracts from the daily journal kept at the Boston office of the Weather Bureau are also contributed by Mr. Smith, and refer to dates which were particularly prolific in heat strokes:

Sept. 5, 1884, "Weather generally clear, and very hot; wind from west or south-west." July 2, 1887, "Very hot and clear; wind west all day." June 13, 1892, "Continued high temperature, very warm, but owing to the low humidity, between forty and fifty per cent., not so oppressive as otherwise would be." June 14, 1892, "Slightly higher temperature and very hot. The extreme heat caused many cases of prostration and sunstrokes." July 25, 1892, "Partly cloudy day, with very humid atmosphere and high temperature." July 26, 1892, "Highest temperature recorded thus far this summer; much lower percentage of moisture in the air (forty-four per cent at 3 P.M." July 27, 1892, "Continued high temperature, less moisture in atmosphere than for several days past." June 6, 1893, "High temperature; numerous prostrations from heat."

The relative humidity, it should be said, is taken regularly but twice a day, so that a true mean is difficult to get at. Still, for our purposes the average answers very well.

In only seventy-three of the cases does the history show whether the heat-stroke was due to direct exposure to the sun or not. Of these, about two-thirds, or fifty-one cases, were direct, and twenty-two were indirect. In the former category were, naturally, many laborers upon the streets and in ditches and sewers. There were half a dozen teamsters, some of whom were stricken while on their wagons. One, it is worth noting, drove an ice-cart. Hod-carriers, coal-heavers, pedlers, masons, carpenters, quarrymen, farmers, etc., made up most of the remainder. Of course, a number of the direct cases occurred in persons not working, but walking on the streets.

Among the indirect cases were three out of the five women. These were employed respectively as cook, worker in a steam laundry, and dish-washer in a saloon. Among the men no indoor occupation proved so liable to heat-strokes

as work in sugar refineries, there being five cases among this class of workmen. Iron-foundry men, bakers, stokers, stablemen, and plasterers were also overcome by heat without direct exposure to the sun's rays. A night-watchman was found unconscious in the morning in his factory, having had to make the rounds of the boiler and engine room, dry-house, etc., in some of which the temperature was known to have been on the night in question 93°.

The following table shows the maximum temperatures recorded while the patient was in the hospital, with the results as regards recovery for each degree of temperature:

Temperature	111 or above.	3	deaths.	0	recoveries.
"	110 — 111	4	"	0	"
"	109 — 110	6	"	6	"
"	108 — 109	6	"	5	"
"	107 — 108	3	"	5	"
"	106 — 107	1	"	4	"
"	105 — 106	1	"	3	"
"	104 — 105	0	"	5	"
"	103 — 104	2	"	3	"
"	102 — 103	0	"	3	"
"	101 — 102	0	"	1	"
"	100 — 101	0	"	6	"
"	below 100	0	"	27	"

The highest temperature recorded in any patient was 111.5° by rectum. The highest temperature in any patient who survived was 109.5°. Both the patients who died having had a temperature of 103°–104°, probably had uræmia, though there was undoubted evidence of insolation being the immediate cause of death. Probably some of the patients who are recorded as having a low temperature (100° or less) had had a considerably higher temperature, which had begun to fall prior to their reception into the hospital.

As to the mortality in general of the 100 cases, 28 died and 72 recovered. All of the deaths, except the two complicated with uræmia just referred to, occurred in cases with a temperature above 105°. Several of the patients were moribund when they entered.

Loss of consciousness was present in 84 of the cases; in 14 consciousness was not lost, and in 2 the record leaves the matter in doubt. In 20 of the 28 fatal cases the patient

never regained consciousness after the initial stroke. The longest duration of such unconsciousness was 30 hours. In 4 or more a partial return of consciousness took place, and in the remaining 3 or 4 cases consciousness fully returned, but not permanently.

The deaths, as a rule, took place within a few hours; but 11 of the 28 fatal cases lived 24 hours or more after the seizure. The longest survival was more than 3 days, and here the temperature had fallen, the patient had regained consciousness and was mending when the delirium returned. He had had a sunstroke two years previously. See Chart IV.

The longest period of unconsciousness in a patient who recovered was 36 hours. Moreover, this person did not enter till the day after his seizure. So that the entire period of unconsciousness seems to have been two days and a half. Ten and 14 hours of unconsciousness with ultimate recovery also occurred, but usually the favorable cases did not remain more than 2 to 4 hours without regaining their faculties.

Convulsions were a very frequent symptom. They are spoken of in 30 of the cases, and were present in at least 13 of the 28 fatal ones. They were tonic and clonic, sometimes local, again general. Universal rigidity and opisthotonos were at times present. Occasionally the convulsions were very violent. A curious spasmodic action which was seen in at least two of my own cases was the rapid darting out and withdrawal of the tongue. Unilateral spasm was occasionally observed. In a patient of Dr. Forster's there was noted "three minutes after death a slow bilateral rotary motion of the arms, beginning in the left and followed by the right. The upper arm was at right angles to body and hands were at shoulders, then both gradually swept down till hands were at umbilicus."

The reflexes were in many of the severer cases greatly diminished or abolished. The pupils were generally irresponsive to light; sometimes they were contracted, and sometimes dilated; often contracted at first, and later dilated; at times they were unequal.

Relaxation of the sphincters and vomiting in unconscious patients were rather frequent.

Albumen was present in the urine in 22 cases, and was

recorded as absent in 32. In about half the cases of albuminuria casts were also found. Whether the albumen and casts had been present before the sunstroke is not, of course, to be known, but in a few instances there is very little doubt that the patient was suffering from Bright's disease when he received the stroke.

The habits of the patients regarding the use of alcohol, like other parts of the previous history, are, as might be expected in patients brought in unconscious, often lacking in the records. Where this point is noted, alcoholic addiction was admitted in probably about the same proportion of cases in which it is found in the general run of hospital patients. In some cases, but not a large number, the individuals had been drinking considerable liquor just before the seizure, and in a few instances the heat-stroke brought on in its train symptoms of acute alcoholismus. The excessive drinking of ice-water is also noted as having preceded the attack in certain cases.

In many of the most serious cases of heat prostration the patients had experienced prodromata of marked significance which should have warned them to protect themselves. For example, the woman already referred to, who washed dishes in a saloon, worked in a very hot place, and for three days had complained of headache and a feeling of lassitude and weakness. A carpenter admitted to Dr. Shattuck's service, with a temperature of 109.4°, subsequently said that for two or three hours prior to his loss of consciousness he had been getting weak, and that objects had looked blue to him. A laborer who was finally prostrated, though not unconscious, said that he had had nausea and great restlessness for four days previously. Headache, faintness, sweating, vomiting, and cramps were other precursory symptoms too often disregarded by patients until too late. A farmer had been working much in the sun for several days, and was finally stricken down by the heat while in a theatre. A laborer felt weak and faint all one day while in the sun, but continued to work; the following day, while driving a cow on the street, began to stagger, but kept on for some time until he fell, unconscious, was brought to the hospital, in

the service of Dr. Rotch, with a temperature of 111°, and died in a few minutes.

The sudden cessation of sweating was noticed by a few patients just before the attack. In one such case a 'longshoreman while "tending hatch" on deck of a vessel at 3 P.M., had been sweating profusely. This suddenly stopped and he "felt cold," then lost consciousness.

The tendency to recurrence of insolation is illustrated by the fact that a history of previous attacks of the same sort was elicited from nine of these patients, and it is probable that in a large number of the patients this point was either not inquired for or not learned. The previous attacks ranged all the way from eight years to a few weeks. One patient, a sugar-refinery workman, was admitted unconscious with heat prostration July 12, was discharged well two days later, was again admitted with a similar attack August 24 following, and again left the hospital "well" in two days.

The treatment followed in most cases was the abstraction of heat by cold baths. These were generally started at 70° or 75°, and rapidly cooled down by the addition of ice. This proceeding is generally followed by a considerable reduction of temperature. Sometimes the chilling of the surface drives the blood in upon the viscera, and collapse occurs. In such cases rubbing of the surface, the application of heaters, and the exhibition of cardiac stimulants is indicated.

The ice-cap and rubbing with blocks of ice are useful supplements to the full cold bath. A cold bath in ice-water sheets proved a useful adjunct. In one very fleshy patient of Dr. Buckingham's, who could not be got into the tub, the temperature was reduced by ice and the external use of ether.

In the earlier years after the introduction of the antipyretic drugs — as antipyrine, etc. — they were used to some extent to combat high temperature in insolation, but this treatment has of late been generally abandoned in favor of the cold baths.

Rarely the temperature rose a degree or so while the patient was in the cold bath. But usually the fall was tolerably prompt. An examination of the thermometric records of the cases generally showed in most of the severer ones a secondary *rise* of temperature coming on a short time after

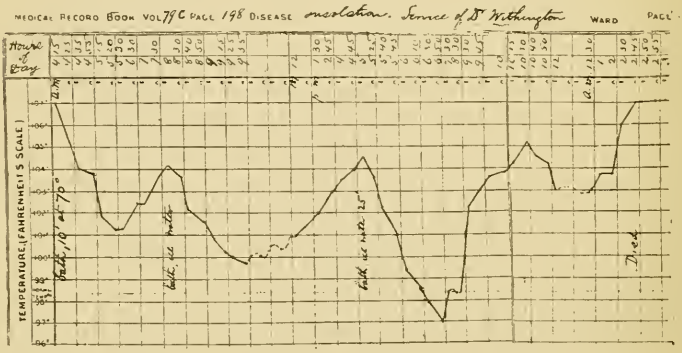
the first fall, and usually not reaching as high a point as the original fever. Sometimes a third or even fourth rise of temperature would occur. The prognosis is grave in proportion to this tendency to subsequent recurrence of the fever. Renewals of the ice-bath sometimes caused a sudden drop of the temperature to a dangerously low figure, 96° or so. Here a copious use of heaters and other treatment against collapse, as referred to above, is urgently indicated.

Of the after-effects experienced by patients following the permanent subsidence of the temperature the chief has been headache. This, with weakness and vertigo, persists, as is well known, sometimes for very long periods. Marked mental delusions were occasionally met with. In two cases mental excitement returned on the second day, and in another, violent delirium came back on the fifth day. This last patient had used alcohol to excess. An acute jaundice followed upon the heat-stroke in one or two patients.

The average period of retention in the hospital for the cases that recovered was exactly four days; that is, omitting one case which developed abscesses from the hypodermic injection of quinine, and was kept for five weeks.

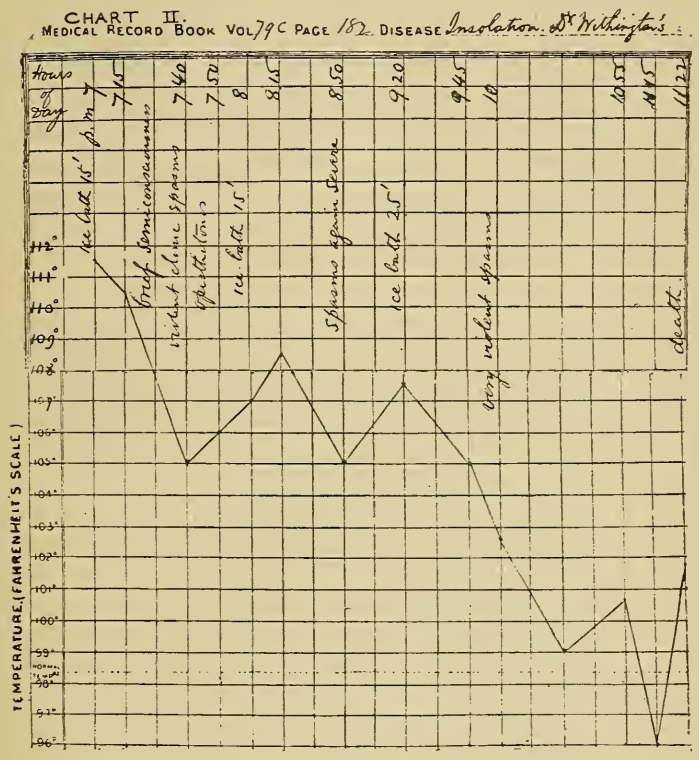
Six of the fatal cases came to autopsy. The lesions found were chiefly subendocardial and pulmonary hemorrhages and hyperæmia of the meninges. Abnormal fluidity of the blood was also noted.

CHART I.



From the clinical histories of insolation I subjoin a few charts to illustrate the graver types of the affection. Case I.

illustrates very well a typical fatal attack of sunstroke, and



especially shows the recurrent failures of the heat-regulating mechanism to maintain itself after the temperature has been

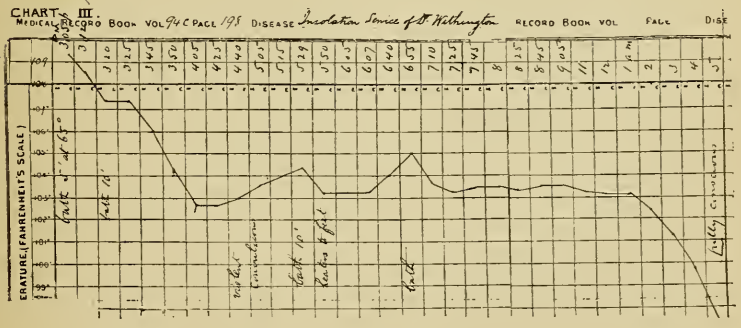


CHART III.
MEDICAL RECORD BOOK VOL 94 C PAGE 198 DISEASE Insolation Service of St Withington
TEMPERATURE (FAHRENHEIT'S SCALE)

reduced by the cold baths. The patient, a laborer, left work at 3 P.M. on July 27, 1892, saying that he did not feel well.

He went to his room, and at 7 P.M. was found unconscious. He remained thus all night, and as he seemed to be getting worse, he was brought to the hospital by the police ambulance, arriving at 4.15 A.M. He entered unconscious, and never recovered his senses more than enough to swallow a little milk. Convulsions were clonic and tonic and frequently recurrent. The chart shows three successive exacerbations of high temperature, and it is to be noticed that the patient finally died with the same temperature, 107°, that he

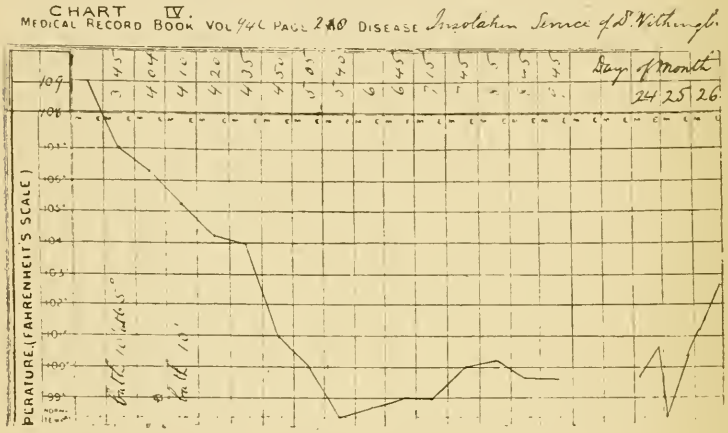


CHART IV.
MEDICAL RECORD BOOK VOL 44 PAGE 288 DISEASE Inebriation Service of Dr. Withington
Day of Month
24 25 26
PERATURE (FAHRENHEIT'S SCALE)

had on entrance. It is further remarkable that the thermometer continued to show per rectum, at five-minute intervals for fifteen minutes after death, the same temperature of 107°.

II. A second case, which occurred nearly simultaneously with the foregoing, July 26, 1892, with the thermometer at 96°, but with a relatively low humidity (44 per cent. at 3 P.M.), showed the phenomenal temperature of 111.5° (rectum), and, as was to be expected, resulted fatally. Its chief features are shown by the accompanying chart, in which the vertical lines represent intervals of about fifteen minutes.

Case III. ended in recovery, and Case IV. died on the third day with a return of delirium after consciousness had been recovered and convalescence seemed established.

IV.

OBSERVATIONS UPON CASES HAVING THE SO-CALLED "TACHES BLEUÂTRES" IN THE CITY HOSPITAL.

By Vincent Y. Bowditch, M.D.

SYNONYMS: TACHES BLEUES; TACHES OMBRÉES; TACHES ARDOISÉES; TACHES PHTHIRIAQUES; MACULÆ CERULEÆ; MACULES CYANIQUES; PELIOMA TYPHOSUM.

Although reference to the literature upon this subject seems to indicate that the presence of these "taches bleuâtres" is of little diagnostic or prognostic significance in any case, yet their precise origin seems still to be a matter of some doubt, and hence a collection of the various theories upon the subject is of interest.

At one time these bluish spots, varying in diameter from the size of a pin-head to that of a fifty-cent piece, not disappearing upon pressure, and most frequently found upon the abdomen and flanks during the course of typhoid fever, were supposed to be due to that disease. Slight allusion is made to them in Flint's "Practice of Medicine," under the head of Typhoid Fever, but no suggestion is made as to their causation. Trousseau had noticed them also in typhoid fever. Other observers found that the spots were not confined to typhoid, but noticed them in various other diseases: yellow fever, pleurisy, pneumonia, gonorrhœa, and also in non-febrile diseases.

In 1868 Moursou, a pupil of Falot, and physician in the French navy, first advanced the idea that these spots were not the result of any one disease, but were due to the presence of pediculi pubis. Later, in quite an elaborate and careful paper, he gave his proofs of this theory, and as a result of his observations makes the following statements:

Over 250 cases had been observed in every variety of climate and in diverse diseases, mild and grave. Delicacy of the skin, a blonde complexion, and a phlegmatic temperament, he noticed were the most usual accompaniments of the spots. He asserts that every time he found these spots the pediculi pubis or their ova were also present. He noticed, moreover, in healthy men who had had the parasitic infection for at least 20 days, that the majority of them had the taches bleues.

Moursou's observations were so carefully made that one is certainly impressed by the probable truth of his theory, which has been substantiated by subsequent observers, viz.: O. Simon, Gibies, Gebbin, notably by Duquet and Mallet. The former injected under the skin a paste made with the bruised pediculi and water, and spots similar to the taches bleuâtres made their appearance. Mallet believed that the color is caused by the secretion contained in the thorax of the pediculus opposite the anterior pair of legs, where there are two pairs of salivary glands, the contents of which are conveyed under the epidermis through the "haustellum" or sucking apparatus of the insect.

Most of the modern text-books in speaking of the "taches bleues" seem to accept the theory of Moursou, notably Ziemssen, Strümpell, and Osler, although Osler evidently feels some doubt as to whether the mere presence of the pediculi is the cause of the spots, or whether there is not some accompanying condition of the patient which favors their appearance. He says: "If really caused by these parasites, it is difficult to understand why they should be present only with fever," which implies that he has not met with them in non-febrile cases. An objection to the theory of Moursou was brought forward by some from the fact that the taches bleues are sometimes found upon people of undoubted cleanliness of body and of the highest moral character; an objection which would not in itself seem to be valid, because of the undoubted fact that such people have often by accidental means become infested by these insects, the presence of which can have been easily overlooked by the examiner.

In an article entitled "Blue Spots in Typhoid Fever," in

the N. Y. "Med. Journal" of Oct. 20, 1894, Dr. A. V. M. Anderson, of Melbourne, Australia, is quoted. In the Australian "Med. Journal" of Aug. 20, 1894, he writes that the "rash" is one of the least commonly seen in typhoid fever, and probably escapes observation on account of indistinctness. He found them in 8 out of 915 cases. In 5 they were present upon entrance, the duration varying from 5 to 15 days; in others they appeared on the 15th, 21st, and 42d days, and disappeared usually in a week or 10 days.

Dr. Anderson could find no evidence of the pediculi pubis, and had not noticed them on patients with other diseases. They were detected over the course of the subcutaneous veins, and would seem to indicate, according to Dr. Anderson, that they are due to an altered condition of the blood, which shows itself in the tendency to epistaxis in so many cases of typhoid. He regards them as of little clinical significance.

Still another theory is advanced by Prof. Revilliod, of Geneva, for the purport of which I am indebted to Dr. C. G. Cumston, who informs me that in a clinical lecture upon the subject, Revilliod stated that he believed the spots to be due to the *excrement* of the pediculus pubis, which he believes to be an insect of very cleanly habits, and that during the night he leaves his usual seat at the root of the hair, and deposits his fæces in adjacent portions of the body. I have been unable, unfortunately, to obtain Revilliod's proofs either for this action on the part of the insect, or for his theory of the excrementitious nature of the spots; but it serves to show the diversity of opinion which still exists upon the subject.

Another theory which has been advanced is, that this insect has a special liking for the bodies of patients suffering from certain diseases; but although they certainly seem to have been noticed more frequently in typhoid fever than elsewhere, as yet no substantial proof has been given that this fact is anything more than mere coincidence.

During my service in the last two years I have come across nine cases in which the taches bleuâtres were present, and in every case the presence of pediculi pubis was proven. Three of these cases were typhoids, which recovered, and the

others were one each of phthisis, jaundice from gastro-duodenitis, acute bronchitis, intermittent fever, hysteria; the one which showed the most numerous spots being a fatal case of cerebral meningitis following an otitis media. Of these cases seven were males and two were females.

In Dr. George B. Shattuck's service this year, up to the present eight cases are recorded in which the blue spots were noticed, but unfortunately no record is made as to whether the pediculi were also present or not. In two which I examined I was unable to find the insect by most careful search, but in one of these cases I only made the examination on the day of discharge of the patient, who had been ill several weeks with typhoid; and in the other the patient had been transferred from the surgical ward as a possible typhoid case, having entered with suspected appendicitis, so that sufficient time had elapsed for the removal of possible pediculi before I saw the patients. The other cases, six in number, were all typhoids, which also recovered.

Judging by the frequency with which pediculi were found to be present in my cases, I can but feel that they would have been found in at least the majority of the other cases had special attention been directed to this point earlier.

Although from such a small number of cases nothing can be proven absolutely, yet they at least show that the spots can appear in quite a variety of diseases, and are not confined to either severe or mild forms of disease.

From this comparatively slight experience I am led to accept the view of Mourson as the correct one.

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V.

A CONSIDERATION OF SOME OF THE INDICATIONS FOR OPERATION IN HEAD INJURIES.

By William N. Bullard, M.D.

The subject of the indications and contra-indications for operations in head injuries is so large that it will be possible to cover only a small portion of the ground in these pages, and I shall therefore confine myself to presenting certain points which seem to me to have not yet been appreciated at their full value by the profession. For the purposes of this paper it will be necessary to limit myself to the consideration of some of the indications for *immediate* operation in cases of head injuries in *adults*. By immediate operations we understand such as are performed within at least twelve hours of the time of the injury. As a rule, the operations under consideration are to be performed within as short a time after the injury as possible.

The indications for later operations, that is, more than twelve hours after the injury, differ very considerably from those for immediate operations, and are, on the whole, much easier for determination. These we will not consider here.

The symptoms on which we rely in order to decide on the advisability of immediate operation in cases of severe head injury may be roughly divided into four groups: (1) extracranial or cranial local symptoms; (2) intracranial local symptoms; (3) extracranial general symptoms; (4) intracranial general symptoms.

(1) The direct local symptoms, those which are immediately due to the injury and are perceptible to the surgeon. Such are wounds, ecchymoses, evident fractures of the cranium, and all other local and evident conditions due to

direct injury in any part of the body, which have any bearing on the diagnosis or prognosis. These are *local extracranial* or *cranial* symptoms.

(2) The intracranial or indirect local symptoms. In this group I would include all localized symptoms which are to be referred to intracranial conditions. Such are the various forms of paralysis, partial or total, of local spasms and certain conditions of the pupils. Symptoms of this group point more or less distinctly to affections of special portions of the intracranial organs, and so far as they do so are *localizing*.

(3) The third group is a somewhat indefinite one. It includes such general symptoms and conditions as may exist after any injury without special relation to intracranial conditions. Under this heading I should place the general strength or weakness of the patient and the condition of the pulse. I have called the symptoms of this group the *extracranial general symptoms*.

(4) The intracranial general symptoms. This includes all general conditions presumably due to intracranial conditions. Such are the states of cerebral activity or of cerebral repose; consciousness, semi-consciousness, unconsciousness, coma, and delirium. These may be reasonably referred to intracranial conditions caused by the injury, and may be fairly considered as the *index* of these conditions.

The direct external symptoms are too well known to every surgeon to render their discussion valuable. The localizing symptoms, though often of great value in combination with other symptoms, in themselves rather determine *where* we shall operate than *when* we shall operate.

I propose to pass over the symptoms which I have placed in the first three groups, and devote myself principally to the consideration of those general symptoms indicative of intracranial conditions which we have mentioned in the last group.

It is largely from the general mental condition of the patient, meaning by this term those states or conditions which we have before enumerated,— consciousness or unconsciousness, delirium, quiet, or irritation, — that the indications or counter-indications for immediate operation in head injuries are in many cases to be drawn. We will now,

therefore, consider the indications for and against immediate operation in cases of severe head injury in adults where the external signs and the localizing symptoms do not exist or do not afford sufficient indication.

The *primary* indication for immediate operation in severe head injuries is *increased intracranial pressure*. The question of operation in these cases depends on the degree of this pressure.

Whenever it has reached a certain point operation is imperative, unless otherwise contra-indicated. This point is the condition of deep stupor where the patient cannot be roused by supraorbital pressure. When, however, the unconsciousness is deep, but the patient can still be roused by passive movements or in other ways, the indication is less definite. Yet in all such cases (and they form a large proportion of those entering the large hospitals) we must be largely guided in our action by the depth of this unconsciousness.

Cases of this kind, as a rule, grow more unconscious during the first few hours; so that if we wait we may often be obliged to act rapidly later. On the other hand, the lighter cases of unconsciousness, which do not tend to become more unconscious, should not be operated upon at once. In any doubtful case the most careful watch should be kept for any increase in the degree of unconsciousness, and as soon as this has been thoroughly determined, operation should be performed.

In all cases, whether only lightly or deeply unconscious, where there has been a rapid increase in the depth of unconsciousness, or where there is a distinct increase of paralysis of the extremities within the course of a few minutes or hours after the injury, operation should be performed. Gradual increase of paralysis, and gradual increase of unconsciousness, becoming apparent shortly after a head injury, suggest intracranial hemorrhage, usually middle meningeal, and demand immediate surgical interference in all persons under forty years of age. In persons much above this age, in cases where the external injury is slight or doubtful, the question of the presence of slow hemorrhage from some of

the deep vessels of the cerebrum (ingravescent apoplexy) must be considered.

When, together with unconsciousness, there exists cerebral activity or irritation, as evidenced by delirium, it has usually seemed to me more advisable to wait and not operate at once, although some of these cases have fatal terminations. On the whole, I consider delirium as rather a sign for delay than for immediate action. Where delirium without unconsciousness exists, it contra-indicates operation or indicates that we should not operate at once.

Localized cerebral irritation, as evidenced by clonic convulsions, either general or local, is not a common sign of head injury (surgical). In cases where there is no history, convulsions render it probable that the case is non-traumatic, or that some ordinarily non-traumatic condition has been set up by the trauma. These are the results of clinical experience.

It would seem scarcely needful to state that in each individual case we must consider the special symptoms and indications. In those cases in which there is no evidence of paralysis, and no pupillary symptoms exist, we rely on the general condition of the patient. The important signs are two: (1) the depth of unconsciousness; (2) the increase of unconsciousness.

(1) It is hard to define exactly the degree of unconsciousness at which — there being no special contra-indication — operation is absolutely demanded. In most of the more doubtful cases we have some secondary symptoms (fracture, paralysis, pupillary conditions) to guide us. Where these do not exist, we must rely to a certain extent on the general appearance of the patient, his pulse, apparent strength, and respiration. If his strength is failing, but he can still bear operation, operate at once. As a rule, we should advise operation in any case where the patient (adult) could not be roused by supraorbital pressure and where the pupils did not react to light.

(2) When the unconsciousness, light at first, rapidly becomes deeper, especially if this be accompanied by any commencement or increase of paralysis, we should at once suspect intracranial hemorrhage, usually middle meningeal.

Where an unconsciousness, deep from the beginning, becomes slowly and very gradually deeper, without an increase of paralysis of the face or limbs, we can only diagnosticate an increase of intracranial pressure, and cannot determine whether such increase be due to hemorrhage or not.

What, now, are the pathological conditions existing in these cases? I believe that wherever the conditions of lasting unconsciousness, stupor and coma exist, we have to deal with an increase of intracranial pressure. This conclusion is based on the results of many operations and observations.

The cause of this increase of intracranial pressure is not altogether plain. It is *not by any means*, as is sometimes supposed, *always* a pressure from intracranial hemorrhage. In fact, I am inclined to believe that pressure from this cause is much less frequent than is supposed. It takes a considerable quantity of blood to produce from outside the brain substance an active pressure on the brain, even when the pressure is produced rapidly.

In many cases, moreover, on operation we have no evidence suggestive of any severe hemorrhage, and yet the increased pressure is apparent. Again, this increased pressure, in all probability, occurs in the cases of so-called concussion, and in other mild cases where unconsciousness exists, but where there can be no question of any profuse hemorrhage. What seems to occur is this: The brain in some way acts as a sponge, and swells and pushes so hard against the dura as to inhibit or diminish pulsation. If in these cases the dura is incised, the cerebral pulsation again becomes visible, and the relief to the patient is instantaneous and extraordinary.

And here let me state that after a tolerably large experience in these operations, I have never seen a case in which the incision of the dura has caused the slightest injury to the patient, or has apparently caused any untoward symptoms whatever.

The cause of this brain swelling I will not discuss here, because it is a difficult subject, not well understood, and, moreover, has immensely broad bearings in various direc-

tions. It occurs undoubtedly in certain cases of apoplexy, and, in a chronic form, in many intra-cranial diseases. This or something analogous, the so-called acute edema of the brain, is the immediate cause of death in cases of acute alcoholism, of sunstroke, and perhaps (in its chronic form) in uremia.

Let us now pass to certain surgical considerations in operations such as we are discussing. It is evident that unless the operation be of a suitable and efficient character, its value is likely to be much impaired and the result not conformable to our expectations. It is as important to know *how* to operate as it is *when* to operate and *where* to operate. It may seem a little presumptuous for one who is not a practical surgeon to attempt to speak on this subject, but I have for years followed these operations with care, and feel justified in laying down certain rules. These are:

(1) Be sure that the opening in the cranium is sufficiently large. In the early days of these operations, and to a certain degree even to the present time, there has been a tendency on the part of the surgeon in exploratory trephining or opening of the cranium to make his aperture too small.

This is a serious matter, both because it prevents the surgeon from seeing and from working inside the cranium at his ease, and still more because it prevents him from opening the dura sufficiently, and efficiently relieving the intradural pressure. I believe that in the ordinary case nothing smaller than a one-inch trephine should be used, and then the opening enlarged, with bone forceps, if possible. An opening, as a rule, to be efficient should be at least two inches by one. Of course it makes no difference how this opening is made, providing that it is made as *quickly* as possible, with as little jar as may be to the patient and without laceration of the dura.

(2) The second point to be emphasized in these operations is always to open the dura where there is evidence of intradural pressure. There is no more danger in opening the dura under proper antiseptic or aseptic precautions than there is in opening any other serous cavity. The superstition that the dura was a structure which it was exceedingly dangerous to meddle with dies hard, and still lingers con-

sciously or unconsciously in the minds of many surgeons and physicians. I can only repeat, that having closely observed for a number of years a very considerable number of patients in whom the dura has been opened, either intentionally or otherwise, I cannot recall a single case *in an adult* where any essential harm was produced. In a very large proportion of cases there is neither any perceptible shock at the time, nor are any future harmful effects (of no matter how slight a character) perceptible.

(3) Remember that in these operations time is an important factor. Do what has to be done as rapidly as possible. In children many deaths are caused in these operations by loss of time. In adults the consequences are not, as a rule, so serious: nevertheless, next to asepsis, time is probably the most important factor in the success of an exploratory cranial operation.

(4) In an exploratory operation, where it is not certain that a large extradural clot exists, look out for the middle meningeal artery. Remember that it runs in a deep groove on the inner surface of the cranium, and that in trephining directly over it, unless the dura be separated in some way from the cranium, the artery is likely to be cut by the trephine before the bone has been completely sawn through. Again, remember how closely the dura is or may be attached to the inner surface of the cranium, and that in cutting with bone forceps in the course of the artery or its branches, great care must be taken not to tear or cut the blood-vessel.

(5) It is perhaps scarcely necessary to mention that all cases of intracranial hemorrhage with which the surgeon has to deal (that is, all cases from superficial vessels, from sinuses, etc.) can be stopped by pressure. Ligating the vessel when possible is preferable, but sometimes it is not possible.

(6) The last point I wish to speak of is a somewhat doubtful one in my mind, and I only desire to call attention to it. This is the length of time during which packing should be left in the intracranial cavity. I am inclined to think that the present tendency is to remove it too early.

VI.

CASES OF PREGNANCY COMPLICATED BY HEART DISEASE.

By George G. Sears, M.D.

Cardiac lesions as a complication of pregnancy have not as yet received the attention in text-books which their importance demands, nor have the best methods of treatment in this condition been adequately discussed in them. This oversight has been partially corrected by monographs upon this subject, as well as by a number of papers which have appeared in the periodical medical press within the past few years; but as their conclusions have been chiefly drawn from cases which presented either very serious or unusual features, the gravity of the complication seems to have been, from the point of view of immediate results, as much over-estimated in the latter as under-estimated in the former. It is not an unusual experience in out-patient work for women to apply for relief of cardiac symptoms, whose lesions were undoubtedly present during one or more pregnancies, and yet were the cause of no serious manifestations, or were even unsuspected by both the patient and her medical attendant. A number of these cases have been selected for a brief report owing to the fulness of the records in several important points, including two which are given in somewhat fuller detail, who sought advice during the early months of pregnancy, and whose course has been followed up to the present time.

Case I. — Mrs. P., thirty-five years old, pregnant for the fourth time. Except for vague pains in muscles and joints no rheumatic history was obtained and her general health had always been good. The birth of the first child was followed by mania from which she recovered after an illness of three months. The second and third pregnancies were

uneventful, but early in the fourth she began to grow somewhat short of breath, and toward the end of the fifth month she noticed that her feet and vulva were swollen. On examination the heart was found to be laterally enlarged, but its exact limits could not be accurately determined owing to the great development of the breasts. A very rough systolic murmur, loudest at the third left costal cartilage, was audible over almost the whole cardiac area. At the apex the first sound was somewhat muffled, but no other modification was noticed. The second pulmonic sound was markedly accentuated, and a few fine râles were heard at the bases of both lungs. The urine was repeatedly tested, but was always free from albumin. Her diet and mode of life were regulated so far as her circumstances would permit, and digitalis and general tonics were ordered. Her condition, if anything, improved somewhat during the next four months, although the edema still persisted. Labor came on at term and lasted about forty-eight hours, but the early pains were infrequent and so slight that they caused but little inconvenience. Active labor lasted but an hour and a half, and as the head steadily advanced and her condition remained good no interference was thought necessary, but ether was freely given. Except towards the close of labor, when it rose to 74, the pulse remained at 60, but it was soft and easily compressed. After the birth of the placenta it fell to 56. The post-partum loss of blood was normal. An examination of the heart made two weeks later showed the following condition: the apex was in the fifth interspace fully an inch outside the normal line, while the area of cardiac dulness extended two fingers' breadth to the right of the sternum. The same rough systolic murmur was still heard all over the precordia, with seat of maximum intensity over the sternum opposite the third costal cartilage. Toward the apex the murmur became much softer in character and could be followed outward into the axilla. The pulmonic second sound was accentuated and reduplicated. Her strength returned slowly, but she insisted on getting up during the third week. Although complaining of dyspnea in climbing a few steps she remained fairly comfortable during the next three weeks, when she "took cold"

and began to cough, raising thin, white sputum somewhat tinged with blood. Dyspnea so increased that she was unable to lie down. Her expression was anxious and the extremities slightly cyanotic. The chest was filled with medium and coarse moist râles, and there was slight dulness at both bases. The pulse was irregular and weak. This condition persisted for about two months, with occasional remissions or exacerbations, one or two of the latter being so severe that stimulants were given hypodermically. She then began to slowly improve, and is now able to attend to her lighter household duties, but dyspnea is still so marked that the ascent of a flight of stairs is attended with considerable difficulty. The baby at birth was a well-formed and apparently healthy child, weighing seven and a half pounds, but it possessed no vitality and died of marasmus when four months old. The third child alone survives.

Case II. — Mrs. H., aged twenty-eight, had rheumatic fever about a year before the birth of her first child, six years ago, and again about two years later. Her second child was born four years ago, and a few months later cardiac symptoms developed. Between this time and the birth of her third child, last October, she had three miscarriages artificially induced, all under four months. She first came under observation at the beginning of her last (sixth) pregnancy, complaining of palpitation, dyspnea and precordial pain. There was considerable lateral enlargement of the heart, a systolic murmur at the apex transmitted outward, and accentuation of the pulmonic second sound. There was neither albuminuria nor edema. Except for a slight increase in the dyspnea and a little swelling of the feet, she passed through her pregnancy without noteworthy occurrence. She was attended in her confinement by her family physician, who extracted the child by forceps after a seven hours' labor, without ether, delivery being followed by a pretty profuse hemorrhage. Four days later she had an attack of syncope, but without losing consciousness, and during the following week she had several similar attacks. Under a tonic regimen she has slowly improved, the murmur has diminished in intensity, and the heart itself has somewhat regained its tone, but she still complains of dyspnea, and seems to be living at

a lower level than before the occurrence of this last pregnancy.

In both cases the history of previous easy labors, the comparatively slight severity of the cardiac symptoms, and the absence of albuminuria, gave every reason to expect a favorable issue under treatment indicated for uncomplicated cases of failing cardiac compensation, and the immediate result justified the expectation. When, however, cardiac symptoms show a tendency to increase in severity, and the patient's condition begins to cause anxiety, it is questionable how far the expectant plan of treatment is justifiable in meeting a complication which, under such circumstances, is fatal in thirty-five or forty per cent. of the cases. In other complications of pregnancy, like uncontrollable vomiting or eclampsia, where the presence of the fetus is the immediate cause of danger, the necessity for emptying the uterus before the patient's condition becomes desperate is generally recognized; yet in both these conditions recovery, if it takes place, is complete, and there is but one risk to consider, while in heart disease not only is the present danger very great, but, if this be successfully passed, the prolonged strain upon the weakened cardiac muscle is apt to permanently incapacitate the patient in a greater or less degree for any form of active life, and be the starting-point for a chain of symptoms which end in premature death. With two dangers to be considered, a remote as well as a present, it is difficult to understand why the same treatment should not have been considered equally applicable to cardiac complications, unless it be that the induction of labor has usually been so long postponed that death has been wrongly attributed to the operation, when of necessity it was the only result to be expected. Within the past year a prompter resort to radical measures has been urged with more or less emphasis by several writers. Ssolowjew,[1] for example, says that when ordinary methods fail, labor should be induced even in desperate cases. E. Leyden[2] is equally positive, and says that the results are so bad because it is postponed till too late. Allyn,[3] in speaking only of mitral stenosis as a

[1] Petersb. med. Woch., Russ. med. Lit., i. 1894.
[2] Zeitschft. f. klin. Med., vol. xxii. [3] Univ. Med. Mag., April, 1894.

complication, says that the frequent occurrence of spontaneous miscarriage or premature labor is "an indication that it should be induced when dangerous pulmonary symptoms persist in spite of suitable treatment." Webster[1] shows more conservatism in apparently taking the opposite view, when he declares that "the quiescent condition of pregnancy is less dangerous to the heart than the disturbance brought about by the emptying of the uterus," but gives no directions for continuing this quiescent state beyond the ninth month. If he means, as he probably does from a later statement, that when a patient's chances have become desperate, induced abortion may only hasten a fatal result, his position deserves little criticism, but should rather be emphasized, in order that she may be prevented from drifting into a condition where death is almost inevitable, no matter what is done.

So many outside considerations come up when the treatment of this complication is discussed, that the main issue becomes clouded; namely, that labor, so far as its effect on the heart is concerned, is primarily a succession of muscular efforts, whose length and severity, unlike voluntary exercise, can only be approximately estimated at best, and which follows a period in which the strength of an already weakened heart is steadily deteriorating. Unfortunately, the welfare of the mother cannot alone be thought of, since a second life is at stake for whose sake greater chances must be taken; but it is an open question if more consideration has not been given to the child, without benefit to either, than is warranted by facts. It is generally conceded that the expectation of life in children born of women with serious heart lesions is decidedly impaired; but the amount of impairment cannot be definitely given until more facts are at hand, reporters of cases not having sufficiently recognized the importance of giving the fate of the children. Beside the two given above, I have notes of five other cases which are complete in this respect, two of which have been reported in detail elsewhere, and will only be referred to briefly here.

Case III. — Mrs. C., thirty-two years old, suffering from

[1] The Hospital, June 9 and 16, 1894.

double mitral lesion probably following chorea in childhood, has borne six children at term, and miscarried once at the fourth month. Excepting the first, marked cardiac symptoms were present in all the pregnancies. The third child alone survives, the others all dying within a few months after birth.

Case IV. — Mrs. B., thirty-six years old, had mitral regurgitation, probably due to an attack of acute rheumatism following the birth of the third child, but cardiac symptoms were first troublesome during her fifth pregnancy. The child lived but eighteen months. The sixth pregnancy terminated spontaneously at the seventh month.

Case V. — Mrs. M., aged thirty-five, vi-para, mitral regurgitation, had chorea when fifteen years old, and rheumatic fever a year or two later. Marked symptoms of failing compensation appeared first during her fifth pregnancy, although there had been a little edema and dyspnea during the previous four, but not of sufficient consequence to require the services of a physician. The sixth child was born about three weeks ahead of time. The last two children lived but a few months.

Case VI. — Mrs. T., aged thirty-two, iv-para, mitral regurgitation, began to complain of dyspnea shortly after her marriage, eleven years ago; but cardiac symptoms did not become severe until her first pregnancy, three years later. Since then she has had three children, the last being born about one year ago. Attacks of syncope, dyspnea, edema of legs, cough, with sputum occasionally tinged with blood, were present in all her pregnancies, while in the latter part of the third she was unable to lie down. In all, menstruation recurred regularly during the first eight months, and after delivery hemorrhage was always so profuse as to "alarm the doctor." Forceps were used in every instance. The first child is living, but in the last stages of consumption. The second child lived three months, and died of convulsions. The third child, now five years old, is strong and well. The fourth died of some acute pulmonary trouble when two months old.

Case VII. — Mrs. H., aged 30, iii-para, had an attack of rheumatism when twenty years old, and has since suffered

from dyspnea on exertion, which has increased in severity since the birth of her first child about four years ago. The first pregnancy passed without noteworthy increase in symptoms until two or three weeks before the birth of the child, when she had a severe paroxysm of precordial anxiety and pain with loss of consciousness. These paroxysms have recurred at irregular intervals since.

The labor was very long, but terminated spontaneously, and was followed by a hemorrhage. The baby lived but fifteen minutes, the only assignable cause of death being the length of the labor. During the second pregnancy there was no increase in the dyspnea and no edema; labor was short, and the child is still living. She again became pregnant last spring, when the cardiac symptoms increased in severity and were accompanied by vomiting. As in the last case, the menses, though scanty, recurred regularly and led to a mistake in diagnosis on the part of the attending physician, whose efforts to relieve a congestion of the womb resulted in a miscarriage at the fourth month. At the time of examination the heart was found to be somewhat enlarged, and a soft, diastolic murmur was heard at the fourth left costal cartilage transmitted toward the apex. At the apex the first sound was rough.

Leaving out of consideration the four cases of abortion, either criminally or accidentally induced, as well as the child whose death was probably due to a too protracted labor, these seven women were collectively pregnant thirty-one times. Of the fifteen children born before the cardiac symptoms became so severe as to require the advice of a physician, twelve are living and three are dead; while as a result of the sixteen other pregnancies but three children still survive, of whom one has but a few months more of life. The sharp contrast between the two sets is so marked that it can scarcely be due to mere coincidence, and should anything like the same mortality-rate, as seems not improbable, be found in a sufficiently large number of cases for definite conclusions to be drawn, much of the force of the most serious objection to an early resort to induction of labor would be lost.

In a paper on the same subject, published about a year

ago, among other conclusions the following propositions were formulated: that the frequency of miscarriage and the doubtful fate of the child make the welfare of the mother the more important consideration in deciding the question of abortion; that the necessity for inducing abortion is very probable in the presence of grave symptoms in the early months, or of a progressive lesion, or of a history of great danger in the preceding pregnancy; and that when the necessity becomes apparent, the sooner it is done the better. Later experience, both with these cases and with a number of others who applied for relief from cardiac symptoms dating from some previous pregnancy or shortly after labor, but whose records are too incomplete to report here, have strengthened rather than weakened this opinion; but the special points which I wish to emphasize are the necessity for classifying heart disease among other conditions in which the presence of the fetus is the immediate source of danger, in order that in this complication the same chance of recovery may be given to the mother which is accorded her in the so-called uncontrollable vomiting of pregnancy or in eclampsia, and that the remote effects of pregnancy and labor, as shown by impaired cardiac compensation, should receive the consideration which their importance demands in deciding the question of the induction of labor.

VII.

REPORT FROM THE DERMATOLOGICAL DEPARTMENT.

By George F. Harding, M.D.

It is a little over twenty-five years since the Dermatological Department was started, and during that period much advance has been made in classification and nomenclature of the various forms of skin disease, although an entirely satisfactory classification has not yet been made.

The list of parasitic affections has been increased, and a number of rare forms of dermatoses have been isolated; many cases formerly included under dermatitis have been shown to be distinct diseases, and given a distinct nomenclature, which, although not entirely satisfactory in every case, serves to distinguish them.

New remedies have been introduced, many of which have proved to be of great value. Pastes have largely replaced ointments in the treatment of many cases. These are made with a starch, dextrine, or gelatine and glycerine base, and are more adhesive and protective than ointments. Unna introduced the so-called "plaster mulls" and "salve muslins," which are useful in some cases, their expense being their chief objection. Icthyol and resorcin have proved of great value, the former especially in erysipelas; and also thiol, which has much the same action as icthyol without having its disagreeable odor, hence it is often to be preferred for use about the face.

Of the various substitutes for iodoform none seem to have proved of particular advantage, except europhen, which Eichoff declares to have a more rapid healing-power than iodoform, besides having the advantage of freedom from odor and absence of risk of poisoning. It is claimed that the drug gives excellent results in burns, both mild and

severe, and Gottheil claims a result with it in six cases of psoriasis as satisfactory as with chrysarobin.

Resorcinol, a compound obtained by heating equal parts of iodoform and resorcin, has acted very favorably in my hands on chronic ulcer of the leg. It has the advantage of requiring but a small quantity, as it should be mixed with starch or talc. It may also be used in an ointment.

Biëlayen says this preparation immediately calms pruritus, but with its use in that affection I have had no experience. I have, however, found an ointment of resorcin very effective in some cases of pruritus and eczema ani. Berger, of Krementchüg, claims to have obtained immediate relief of pruritus ani by the use of liquor calcis chloratae (B.P.). A plug of cotton soaked with this liquid is inserted about an inch into the anus and allowed to remain until a slight smarting sensation is produced; the plug is then removed, and the whole region is washed with the same solution, which is allowed to dry on.

The use of thyroid extract in the treatment of certain forms of skin disease does not seem to have proved of any advantage over the present methods of local treatment, while on the other hand it has the disadvantage of producing constitutional symptoms which are disagreeable and at times dangerous to the patient.

Unfortunately, since there are no beds in the hospital at the disposal of the department, it is impossible to test the effect of many of the new methods of treatment. The patients do not apply the remedies properly at home, nor can they come daily for observation, which is quite necessary in testing new drugs, owing to the difference in susceptibility of different skins. Again, many of the new drugs are too expensive for the patients to buy.

During the past year I have had an opportunity of observing some unusual cases among those who attended the clinic, and I present them here in brief, as, unfortunately, I have but little of their individual history.

As a result of the universal vaccination of last year, a number of cases came to the clinic. Some of these were simply "bad arms" which refused to heal; others showed a margin of erysipelatous inflammation; while others showed

an impetigo contagiosa, which is the most common of the post-vaccinal eruptions. Two cases showed a generalized scarlatiform eruption, which appeared about ten days after the vaccination, and corresponded to the so-called roseola vaccinia.

The three following cases belong to the more unusual sequences of vaccination, and I give them a little more in detail:

Case I. — M. L., a strong, well-nourished girl, fifteen years of age, presented herself, with a well-marked general psoriasis. She had been vaccinated about four months before for the first time. The vaccination took, and she had a very sore arm. Before the pock had healed she noticed some small "pimples" around it. These became scaly, increased in number, and extended up and down the arm, and thence to the back. Some time after this it was noticed through and around the borders of the scalp, and later some few scattered spots came out on the front of the trunk, on the legs, and on the other arm. She had never had any skin disease before, nor had any of her family. Vaccination in the other members of the family took the usual course without any after result.

Case II. — M. C., twenty-three years of age, was vaccinated on the left arm in two places. After four or five days, the vaccination having taken, he noticed a severe neuralgic pain in the neighborhood of the pock, which was followed by the appearance of "pimples like blisters." These continued to appear, and when I saw him he had a well-marked herpes zoster, extending from the pock over the deltoid region. A week later a patch appeared in the intercostal region. Previous to vaccination he had felt perfectly well.

Case III. — M. D., nine years of age, had had no skin trouble and was in good health previous to vaccination. Her mother stated that she was quite sick after the vaccination, and had a "bad arm." She did nothing for her, however, till about ten weeks after, when she noticed an eruption coming out over the body, which took the form of blisters and itched badly. This was treated with various preparations obtained from the drug-shops and a physician whom she consulted, but, as it continued to increase in severity,

she finally came to the hospital, hoping to get the child in there.

When I saw the child she presented a perfect picture of During's dermatitis herpetiformis. The whole body was covered with vesicles varying in size from a mustard seed to a small bean. In some places the vesicles had ruptured and left dried crusts, while almost all the body was marked by excoriations caused by scratching, which she kept up continually during the examination.

Whether these cases can be ascribed directly to vaccination or not, it is difficult to say. Certainly a large number of eruptions do occur after vaccination, and several cases of post-vaccinal psoriasis have been reported. It is possible that the exciting cause may be introduced with the vaccine virus, or cases like those mentioned may come under the head of dermato-neuroses, and be caused by peripheral nerve irritation.

There were three cases of urticaria visiculosa, all in children under four years of age. This is one of the less common forms of urticaria, and resembles closely the early stage of dermatitis herpetiformis. It is more amenable to treatment, however, than that disease, and not so chronic in character. It is caused by a superabundance of serum in the wheal, which, forcing its way through the rete, elevates the upper layers of the skin, thus forming a vesicle on top of the wheal. In all these cases the eruption disappeared under the influence of a mild antipruritic lotion and attention to diet.

There were three cases of prurigo, two of the mild form, prurigo mitis, and one of the severe form seldom seen outside of Austria, prurigo ferox. For a long time the existence of prurigo in this country was denied, the cases which were reported, and which were of the mild type, being considered by many as a papular eczema. A sufficient number of cases have been reported, however, to show that the disease does occur in this country, and is not remarkably rare in the milder form. In the severe form only a few cases have been reported; and as the following case was a rather typical picture of this form, I give as full a sketch of it as my few notes will permit.

M. W., twelve years old, showed an eruption on the legs and arms confined most markedly to the extensor surfaces. The eruption was of a papular nature, accompanied by deep infiltration of the parts, and excoriations caused by constant scratching. There were also crusts, formed by the dried blood and serum, and pigmentation, chiefly about the thighs. The glands in the groin were enlarged and indurated, as were those of the axillary region, but to a lesser degree. The child was extremely cachectic, and had never been well or strong. The mother stated that the disease had begun when he was about a year old with "itching spots" on the legs, which increased in number, and finally appeared on his arms. The present condition had existed for about five years, during which time the child had been running down more and more, and, in spite of various forms of treatment which she had tried, had shown no improvement. The disease had never shown itself on any other parts than those named above. She had other children, but none of them had had any form of skin disease.

An interesting case of dermatitis was that of a girl who worked in a chocolate manufactory, her duty being to stir the hot chocolate. She presented on the flexor surface of the right forearm, extending half-way up it, a papulo-pustular eruption, which she said had existed about a year, during which she had been employed, as above stated, in the chocolate manufactory. There was a slight itching at times, but otherwise no subjective symptom. She was well nourished and otherwise in perfectly good health. I ordered a zinc paste, and advised her to change her work, which she did, going into the packing department, and in about a month the skin was in a normal condition. She now returned to her old work, and shortly after appeared with her arm in the same condition as formerly.

The condition of pompholyx, or cheiro-pompholyx as it is called by some, was shown in two patients, one a man eighteen years of age in whom it had existed for a month, the other a girl of seventeen who had noticed it for three days.

This is a disease which shows itself in the form of vesicles or bullæ, limited to the hands and feet. It is said always

to appear on the hands, while the feet may be quite free from it. In the two cases mentioned it was limited to the hands. The eruption appears as a rule along the insides of the fingers, or on the palms of the hands or soles of the feet, but the whole surface of the hands and feet may be affected. It shows itself at first as vesicles, which are said to resemble sago-grains imbedded in the skin. These later become raised above the surface in the form of bullæ, sensations of tingling and burning accompanying their development. After the lesions have fully developed, the fluid becomes absorbed, the skin sinks down and exfoliates, leaving a red denuded surface. It seems to occur in persons who are run down, and, according to Crocker, is seen chiefly in the summer. Neither of these cases, however, occurred during the summer, but both stated that they had not felt well for some time.

In both of these cases, as I have said, the eruption was limited to the hands. In the girl it covered the hands, back and front, while in the man it was only on the palms and between the fingers, some denuded spots showing where absorption and exfoliation had taken place.

Fred. F., a boy thirteen years of age, showed a forefinger encircled by a well-defined band of deep-red color, which covered the second and third phalanx. His mother stated that it started, two weeks before, as a red spot on the tip of his finger, from which it had extended gradually to its present limit. It was attended by a burning sensation, and at times by itching. He was, and had been, perfectly well, and suffered no other inconvenience than the local symptoms already mentioned. The surface was free from scaling, and had never shown any tendency to become scaly.

This condition corresponds to the erysipeloid of Rosenbach, described by Elliot in Morrow's "System of Dermatology." According to Elliot, it develops around the point of infection, which only need be a scratch or insignificant wound, in the form of a slightly elevated, well-marked zone of dark-red color. Thence it extends peripherally, accompanied by sensations of pricking, burning, and itching, the old portions fading as the new line progresses. The progress is very slow, and a portion of the periphery may

become normal while the extension continues in another part, thus giving a serpiginous border. As the redness disappears, a yellow color takes its place, but at no time does desquamation or scaling occur.

It occurs chiefly on the fingers and hands, and is most common among cooks, butchers, and people of that class who handle meat, fish, etc. Rosenbach claims the cause to be a distinct micro-organism which exists in dead and decomposing animal matter. Elliot cites a case in which autoinoculation occurred, although it is claimed that the disease is not contagious.

Richard C., eight years of age, presented a patch of lupus on the scalp, in the region of the vertex, about the size of a silver dollar, which his mother said had begun when he was about three years old. There were no other patches on the body, nor any remains of such in the form of cicatrices. None of the other children in the family had shown anything of the kind.

The tissue was soft and boggy, allowing a stick of nitrate of silver to penetrate easily, but showed no tendency to ulcerate.

I have mentioned this case because of its extreme rarity. The majority of the cases reported reached the scalp by extension of the disease from the face, and one author denies the existence of primary lupus of the scalp. To my mind there is no doubt as to the diagnosis in this case as the nodules were distinctly visible, and the tissue was boggy and soft. The slow progression of the disease and the age at which it first appeared are also strong evidences in favor of this diagnosis. In other respects the child's health was good, and always had been, and he appeared well grown for a child of that age.

There are always a large number of cases of impetigo contagiosa and eczema about the face in children, due primarily to pediculosis capillitii. It seems to me that the number of these cases could be largely reduced if some attention were paid to the subject in the public schools, where the opportunities for contagion are very large; and now that medical examiners have been appointed for each school, the problem does not seem to be a difficult one. It

is not necessary to keep such children from school, for the treatment, which is generally a matter of but a few days, can be carried on either at night or between school hours. It is, however, necessary for the parents to be told to look after their children's heads, and see that they are properly washed with soap and water at least once a week. Another aid would be in seeing that each child keeps its own headgear to itself; for I find that it is not uncommon for these children to interchange their hats, and when the woolen "Tam O'Shanter" is worn, as it seems to be pretty generally, it is almost impossible for contagion not to occur. I believe it is by this means that many children of the better class obtain their unwelcome visitors.

Another class of cases, which we are less able to control, occurs in infants under ten months of age who are fed on certain of the prepared foods which contain too much sugar. I have seen a number of cases of furunculosis in infants who were fed on condensed milk, and in whom the eruption ceased shortly after the food had been changed to properly prepared cow's milk.

A number of cases of fugitive eruptions have occurred in the house, in which it was almost impossible to give a distinct diagnosis, other than dermatitis. Under this head come the long list of drug eruptions in which the cause can only be maintained when it is known that the drug has been given; and when several drugs have been given it is difficult to say, in many instances, which was the direct cause, since many of these drug eruptions are of the same type. For example, no less than twenty-three drugs are reported as having produced an erythematous eruption. Then there are the several forms of erythema, and the rashes at times accompanying or preceding certain general affections.

Desquamation may follow almost any of these rashes, and in some cases there may be a general systemic disturbance accompanied by a rise of temperature. In erythema scarlatiniforme there is often a distinct prodromal period followed by lassitude, and fever lasting two or three days, which renders its distinction from an exanthema difficult for the time being. Add to this the fact that desquamation almost always follows to a marked degree, and it becomes difficult

to judge whether the case were not one of scarlatina, which in consequence should be isolated.

Some cases of variola are preceded by an erythematous eruption which is most marked about the axillary, hypogastric, and pubic regions, and the inner sides of the thighs, and which fades almost entirely before the variola eruption begins.

An erythematous eruption may appear at the beginning of, or during, an attack of diphtheria. Sometimes it appears only on the trunk, while at others it shows itself over the whole body and closely resembles scarlatina. Its appearance in the later stages is regarded by some as due to toxic invasion.

There is also a form of erythema which may appear in children and persons with delicate skin during high fever from any source. Rashes may precede or occur during an attack of cholera or cerebro-spinal meningitis, which are for the most part of the erythematous type. In one case of the latter which I have in mind, erythematous patches of a bright-red color, about the size of a silver dollar, appeared over the face and trunk the day before the patient died.

In septicæmia a scarlatiform general eruption is of frequent occurence, and in uræmia eruptions of the same type have been observed.

The following case of digitalis dermatitis, which occurred in the service of Dr. Monks, to whom I am indebted for the notes of the case previous to my seeing it, is interesting in this particular.

F. H., aged thirty, was admitted to the hospital on July 27, with a history of not having urinated for three days. He also complained of pain over the sternum, and hiccough. Physical examination showed a stricture of the urethra through which no sound could be passed, and catheterization was attempted without result. Percussion showed that the bladder was not full. July 28, two filliform bougies were passed, and the patient received ten minims of tincture of digitalis every four hours. During the night he passed half a cupful of urine, the bowels moved, and there was some vomiting. From this time on the amount of urine passed showed a steady increase.

August 1, *i.e.*, four days after the digitalis was first given, an eruption was noticed on the trunk, composed of discrete papular lesions of a bright-red color. August 3, when I was called to see the patient, there was an eruption of fine papules of a dullish-red color spread over the trunk and arms in confluent patches, which disappeared under pressure, and which left small areas of normal skin between them. On the face and neck were a few scattered patches of the same color, but rather more distinctly papular in character. The lower extremities were practically free from any sign of the eruption.

The dose of digitalis was now reduced to ten minims three times a day, and on the following day it was omitted. The eruption began to fade on the day after the digitalis was reduced, and four days later, when I saw the patient again, there was but a faint trace of it. Whether desquamation followed I am unable to say, as the patient was discharged, at his own request, as soon as the eruption had faded.

In attributing an eruption to digitalis in a case like this, one must take into consideration the possibility of an uræmic erythema, of which I have spoken above, and which closely resembles in character this eruption. On entrance the patient showed some signs of an approaching uræmia, but these had entirely disappeared at least two days before the eruption showed itself. Hence it seems to me that the eruption can fairly be attributed to digitalis, especially as the patient had received no other medicine.

Cases of digitalis dermatitis have been reported by Traube and Morrow. One of Traube's cases is that of a patient with pleuro-pneumonia and acute nephritis who was given three grams of digitalis, in infusion, in three days. On the third day a redness of the face was noticed, which increased on the next day, one gram more having been taken. The digitalis was then stopped and carbonate of ammonium given. After using this for three days, a red papular eruption appeared on the trunk, forearms, and backs of the hands, spreading next day to the neck and upper arms, and becoming more confluent on the trunk. Desquamation

occurred on the face, but the eruption on the trunk and extremities disappeared in four days without desquamation.

In Morrow's case, mentioned in his book on drug eruptions, the patient had been taking infusion of digitalis and acetate of potash for suppression of urine. An erythematous eruption resembling scarlatina appeared after a few days, and on the following day, the medicine being continued, an urticarial eruption showed itself. Shortly after the medicine was stopped the eruption began to disappear. Desquamation followed, with alopecia of the scalp and shedding of the toe and finger nails. Morrow also mentions a case reported by Schuchardt, where an eruption of similar character to Traube's was twice developed by digitalis.

The general character of the eruption in the case which I have reported agrees with those above mentioned, but differs from Traube's case in that it began to fade shortly after the dose of the drug was diminished, whereas in his case it did not begin until several days after the drug had been stopped. Morrow attributes this late development of the eruption to the cumulative effect of the drug.

The alopecia and shedding of the finger and toe nails, which occurred in Morrow's case, appears to me to be a much severer after-effect than one would expect, and I cannot but think that such a result must have another cause than the digitalis.

Leon Perrin has described certain eruptions which may occur during an attack of gonorrhœa, apart from the balsamic eruptions, but which are comparatively rare. These he says appear about the third or fourth week, on an average, but some months may pass before there are any signs of them.

He classifies them under three heads: purpuric; erythema, multiforme and nodosum; scarlatiniform or morbilliform. The first class are rare, and occur on the lower extremities of persons who are run down. In the erythema form, a constitutional disturbance precedes the attack by a few days, and the eruption, except of course in the nodose form, generally appears first on the neck and trunk, the mucous membrane also being affected.

It disappears in about a week, and may be followed by slight desquamation.

The eruption may be confused with the exanthemata, or the prodromal symptoms may be so severe as to suggest an attack of typhoid fever.

He ascribes the cause to the intervention of the nervous system rather than any microbic or pyæmic influence.

VIII.

THE ULTIMATE RESULTS AFTER ALEXANDER'S OPERATION.

By John G. Blake, M.D.

In the limited space at my disposal in this volume, it has only been possible to summarize briefly the views of my correspondents, and not be over-generous in setting forth my own. The results, as shown by figures, are more convincing than any individual opinion or experience could be. My claim to credit is only that of recorder, with my own slight contribution to the general statistics.

When one has done between fifty and sixty Alexanders, with satisfactory results in the main, and then hears its utility doubted at the meetings of a prominent medical society, the question naturally arises: "Am I deceiving myself and taking too favorable views of results, or are the critics competent, and their objections well founded?"

In common with many other surgical procedures, the limits and conditions necessary for success were neither fully understood nor observed in the early days of its performance. As a natural consequence failure and disappointment often followed. Many became discouraged, and abandoned it altogether; others, looking upon it with disfavor, sought to find a substitute. As a result we have had various methods for ventro-fixature, which, looked at from either the anatomical or surgical standpoint, have not the same claim to scientific justification. It certainly seems reasonable that if a couple of the stays or guy-ropes of a derrick become slack, allowing the structure to sag, the sensible way to bring it back to a normal position would be to haul in the slack and belay them. This may appear to be a somewhat coarse and imperfect illustration of the operation, but it is really what we do in Alexander.

If, in addition, we remove the causes and results of displacement by curetting, we help to restore the organ to a normal condition, and render success more certain. If we give support also by straightening the uterus, keeping it so by means of an intro-uterine stem, and use some form of retroflexion pessary, as recommended by Dr. Alexander himself, we have all the conditions for success — provided the case was a proper one for the operation in the first place. If, however, we take a heavy uterus adherent posteriorly to the rectum, endeavor to lift it up, and succeed only in lifting the parts to which it adheres, without breaking the adhesion; if we do not diminish the weight of the organ by curettage; if we give no backward support, but allow the strain of the adhesion and weight of the uterus to come on the ligaments while the process of healing is going on, and even for some time after completion, — we must not expect anything but failure, and should not be disappointed in the result. The operation was never intended for such cases, never succeeded in them, and never will. Simple retroversion, without adhesion, is its field, or with adhesions that can be thoroughly broken up at the time of operation.

Now I feel convinced that most failures come from non-observance of these primary rules for guidance. I am equally certain that if the critics of to-day would exercise the same care and discrimination, both in selection and treatment, that is shown by the men whose favorable reports are set forth in this paper, they would have equal cause for congratulation. This is the natural inference to be drawn from the statistics. It is perfectly evident that no sane man would continue operating year after year if his results did not justify continuance; neither would patients subject themselves to the restraint and expense, unless encouraged by successful results in others.

In view of the differences of opinion, the fairest and broadest way appeared to be that of gathering a consensus of the profession at large outside the influence of local opinion and prejudice. With this view a circular was addressed to many well-known gynecologists of this and other countries, asking the ultimate results after operation, and the effects of child-bearing on the position of the uterus. The

kind and quick responses coming promptly and fully from so many busy men have filled me with a lively sense of gratitude which I cannot forbear expressing. It will be seen that the largest percentages of good results are with those who have operated the greatest number of times, and who have therefore presumably learned to discriminate in the selection and treatment of cases. In my opinion failure is always the fault of the surgeon, and not of the operation. The careful, painstaking man chooses the proper case, is not confused because he does not find the ligaments readily, or discouraged if they prove to be small in size; is calm if he must go well up into the internal canal to reach them; is careful about the insertion of sutures, and the proper closing and dressing of wounds. Given these conditions, I defy failure, save in the exceptional case. This may appear a strong statement; but it is justified by the experience of others as well as my own.

Having thus briefly — but I think not too forcibly — set forth my own views, let us review the opinions of others. We must remember the difficulty of following up hospital cases, and getting final results with accuracy; but it is fair to assume that patients will seek us out in case of failure; or, if they do not, that we shall hear of them from some brother practitioner. It is to be remembered that the operation does not always succeed in relieving all the symptoms; partly because of the existence of other conditions not fully taken into account, partly from expecting too much at the outset. But the number of these is relatively small; not larger than is found in the records of any well-recognized surgical procedure. I have preferred quoting from the letters of my correspondents, giving their opinion in connection with their figures, as being more satisfactory than a mere tabulated view. Want of space prevents fuller statements. It will be noticed that a wide space is covered — from Dr. Kollock, of South Carolina, to Drs. Tait and Keith, of England.

Dr. Kellogg, Battle Creek, Mich.: "I have performed the operation for shortening the round ligament 328 times (modified Alexander). Some ten or twelve cases, in which pregnancy has occurred subsequently, have been reported to

me. Most of them I have had an opportunity to examine. In all except one the uterus has been found in a normal position. Less than five per cent. of my cases are failures — mostly where the abdominal viscera are prolapsed, with weakness of the abdominal muscles, so that the natural supports of the uterus are wholly lost."

Dr. F. W. Johnson, Boston: "I have on record, at the Carney and St. Elizabeth Hospitals, 250 cases of the Alexander. Twelve have been delivered at term. In every case the uterus has been examined, and was found in perfect position. In no case has hernia followed the operation. Of the 250 cases, eighteen left the hospital with the uterus more or less retroverted. Two died — one of sepsis, and one of pneumonia."

Dr. Henry T. Byford, of Chicago: "Have performed Alexander 50 times. A few have had children, but I do not know how many. A return occurred in a few of my early cases — none for a long time, since I have learned how to choose them. I regard it as one of the most natural and useful operations in minor gynecological surgery. I nearly always use a pessary for a few months."

Dr. W. H. Baker, Boston: "There have been fifty-eight operations at the Free Hospital for Women. Cannot tell how many I have done in my private practice. Am heartily in favor of the operation in properly selected cases — malpositions without adhesions. When catgut was used, malposition was produced in a few months; when braided silk was substituted, the effect has been permanent with one exception. No knowledge of cases of child-bearing after operation."

Dr. L. H. Boldt: "Have operated on fifty cases — one failure; first case. Four have borne children subsequent to operation, and the uterus remained normal in each case. All the other cases have been perfectly successful."

Dr. George M. Edebohls: "Up to the present time I have shortened the round ligament upon ninety-one patients. July, 1893, I reviewed my cases to that time — sixty-two. All were operated upon by a modified Alexander-Adams operation, described in 'New York Medical Journal.' Final result in sixty-two cases, to July, 1893: Sixteen patients

left hospital four weeks after operation, uterus in normal position, and not again seen; three cases, one ligament torn out in attempting to shorten it — in all three ventral fixation immediately performed; one case required hysterectomy for sarcoma about a month after operation. In remaining forty-two cases, uterus was found in normal position when last seen, at periods varying from two to thirty-eight months after operation. Up to July, six of the forty-two cases had become pregnant — one of them twice; seven pregnancies in all to my knowledge. One was advanced to second month, two to third month, when last seen by me; a fourth miscarried at third month. Three went on to deliver full-grown, healthy children at term. All these mothers examined several months after delivery, and uterus found in normal position. Since July, 1893, one case had relaxation of ligaments, and uterus became retroverted. Since that time five or six more have had children at term; one is still pregnant."

Dr. Paul F. Munde: "I have done the operation sixty-eight times. All but two were successful; some few failures. In relation to the test of pregnancy, I have seen four such; one in her fifth confinement, and one six years after the operation. No displacement. Drs. Coe and Boldt have each reported one, in which the uterus remained in normal position."

Dr. Rufus K. Kingman: "Almost fifty, nearly all successful. Some few failures where catgut was used for sutures. No pregnancies that I have heard of."

Dr. J. W. Elliot: "I think I have done the operation about fifty times. I know the ultimate result only in a few cases; several of these were permanently cured."

Dr. Malcolm Storer: "Eleven cases; two subsequent pregnancies, in one of which uterus known to have returned to normal position. Ten successes, one flat failure. Two not relieved as regards symptoms; both hypochondriacs."

Dr. Frank B. Harrington: "Operated forty times; recovery has always been speedy, without serious results. Should say that in about one-third of the cases ligament had stretched. In two of the earlier cases a slight hernia followed, easily controlled by truss."

Dr. F. H. Davenport: "I have done in all about fifty operations. One became pregnant, and miscarried between the third and fourth month; uterus fell back. Only in one other did the uterus return to malposition."

Dr. Edward Reynolds: "I have delivered a number of women upon whom other men had previously done an Alexander, and have never seen one in which any ill result followed. I have never myself seen a malposition return."

Dr. Cornelius Kollock: "Have operated five times. Three have had children, one or more, since. All fine, healthy. Mothers likewise strong and rugged. Slight return of malposition in one of the other two."

Dr. W. F. Burrage: "Have done the operation thirty-five times, with good immediate and remote results in all but one case. As far as has come to my knowledge, only one has become pregnant. Result on uterus unknown to me. In out-patient services at hospital, have seen results after operation; only a few failures, and a great many satisfactory."

Dr. M. H. Richardson: "Experience limited to three cases. No children. First case no benefit; other two did well while in hospital. Have succeeded in demonstrating time and time again on the cadaver that the uterine body can be brought into beautiful position by this operation."

Dr. W. M. Polk: "I have performed Alexander's operation about ninety times. I know of four who have had children, and in three of these the position of the uterus remained after delivery as before. The fourth was a case of procidentia, and after delivery the position of the uterus was as bad as at first. The more I do the operation, the more I find myself in accord with the limitation placed upon it by Alexander himself. It is an ideal procedure for simple retroversion without adhesion. If the adhesion can be directly broken up per vaginam, as I am doing the operation, it can, I believe, be made ideal for retroversion with adhesion. Its utility is questionable in every other state."

Dr. Reuben Petersen, of Grand Rapids, reports six cases, all successes. Three had children at term, without change in the position of the uterus.

The above fairly represent the results of the largest oper-

ators friendly to the Alexander. In my own experience, comprising between fifty and sixty cases, I should say that fifty, at least, were permanently benefited. The others included failures from ill-selected cases, inexperience in the earlier operations, and want of attention on the part of attendants. Three of my cases have borne children; and in all the position of the uterus remained unchanged. The operations were done either at the City, the Carney, or the St. Elizabeth Hospitals.

On the other hand, a large number of men eminent in gynecology condemn it outright, or give but very qualified commendation, mostly on theoretic grounds, or from limited experience.

Among these may be mentioned Drs. J. and M. Price. Dr. Battey, of Georgia, never did the operation, and has not been favorably impressed with it. Dr. Dudley, of Chicago, operated five times with fairly good results, but does not esteem it very highly. Dr. W. R. Pryor, of New York, has never done the operation, preferring to obtain results by other methods; was unfavorably impressed with the results seen by him in out-patient clinics. Dr. Virgil O. Hardon, Atlanta, never operated, because he was not favorably impressed, and it did not commend itself to his judgment. Dr. Walthen, of Louisville, preferred abdominal section, fastening the organ in front, from which he gets satisfactory results, except when he opened the abdomen and shortened the ligaments that way. Dr. King had never done it, and was non-committal. Dr. Theophilus Parvin had never operated. Dr. Grandin, while admitting a limited sphere of usefulness, thinks that it will not long continue to be regarded as a scientific operation, and will die out; regards it as more or less of a fad of the present time. Dr. Martin, of Chicago, believes in the operation under limited conditions, but makes no reference to any done by him. Dr. Engelman, of St. Louis, does not favor it. Dr. Lawson Tait operated twice only; one patient nearly died; the result was a failure in the other, within twelve months. He had also seen failures in the results of operations by others. He had no confidence in its permanency. Dr. Skene Keith had done it but once, and was disappointed in the ultimate result. Dr.

Keith, Sr., fixes the uterus by drawing one broad ligament into the abdominal wound. Father and son have done this operation between fifty and sixty times since 1869, without a single failure.

Many others have responded, but prefer not to be quoted; some on account of the small number of cases, or their inability to look them up and ascertain results, and others because of intention to publish themselves. We have, however, nearly twelve hundred operations, with the estimates of success by well-known, reliable men. The number of child-births, about fifty-three, is conclusive reply to the claim that pregnancy is prevented by it, or that the good results of the operation are destroyed when it follows. It is also shown that abortions are exceedingly rare as a sequence. But perhaps the point best demonstrated is the tendency to disagreement that has become proverbial in the profession, and which the answers to my inquiries prove to be still flourishing in undiminished vigor.

IX.

CARIES OF THE AUDITORY OSSICLES.

By J. Orne Green, M.D.

Chronic suppuration of the tympanum in almost all cases owes its chronic character to the existence of one or more conditions which we can designate as complications, and the relief of these complications will enable us to cure the existing inflammation, and relieve the patient of the otorrhœa and its attending dangers. The recognition and relief of these complications become, then, an all-important factor in diagnosis and treatment. The disease begins as a suppuration of the tympanic mucous-membrane, almost always attended by perforation of the drum-membrane of greater or less extent, and the complications are the direct result of this inflammation, being either retention and decomposition of the inflammatory products, inflammatory hypertrophies of tissue, or extension of the inflammation to neighboring parts. They can be classified as follows:

1. Retained secretion.
2. Desquamative products.
3. Granulations and polypi.
4. Caries and necrosis.
5. Mastoiditis.
6. Labyrinthic inflammation.

The original inflammation produces the complication, the complication keeps up the inflammation. In fact, we have here a vicious circle, and the complication must be removed before the tympanic inflammation can be successfully treated. In every case of chronic tympanic suppuration a most careful search must be made for each and all of these complications by careful inspection, by the probe, and by the study of the symptoms, both subjective and objective.

Caries may be found of any extent, from a superficial spot the size of a pin's head up to a large portion of the petrous bone. It is found in any portion of the bone, in the tympanic ring, on the promontory, at the tympanic orifice of the Eustachian tube, in the additus ad antrum mastoideum, in the mastoid itself, in the tympanic attic, or lastly in the ossicles.

Wherever located, caries will keep the mucous membrane of the tympanum inflamed, and the disease of the bone must be cured before the otorrhœa can be reduced. We find that nature cures the bone in one of two ways, either by throwing off the diseased portion in the form of a sequestrum, or by a slow disintegration of the diseased part, which disappears in the discharge. We often can assist both forms by instrumental interference, removing the sequestrum when it has formed, and saving the time that nature would take to displace it from the mass of granulating tissue in which it is usually buried, or accomplishing by means of the curette in a few minutes the disintegration for which nature might require months or years. In either case we greatly diminish the risks of further involvement of the bone, and reduce the risk to the patient by the time we gain over the natural process.

Caries of the ossicles is particularly difficult of absolute recognition, for the spot of caries may be very limited in extent, so that it readily escapes the probe even when the destruction of the drum-membrane is so great as to allow that instrument to be passed freely up into the attic. Or in many cases the perforation of the membrane is so small that a probe is passed with difficulty and can reach but a portion of the attic.

A carious ossicle becomes in practice an ordinary sequestrum. The cause of caries of the ossicles is undoubtedly the retention and decomposition of the inflammatory products in the upper portion of the tympanum, now called the attic, and recalling the anatomy of this cavity it will be seen how readily this may occur: for in this small space we have the head and neck of the malleus, the body, short process and long process of the incus, the stapes at a right angle to the incus, the stapedius muscle crossing the cavity in one direc-

tion, the tensor-tympani muscle crossing it in another direction, the chorda-tympani nerve running between the two larger ossicles, the suspensory ligaments of both malleus and incus, and finally the reduplications of the mucous membrane, which covers all of these structures, and which when slightly swollen from inflammation completely closes this upper space from the rest of the tympanum. A more unfavorable condition for drainage and thorough surgical cleansing can scarcely be imagined than this cavity shows in its natural conditions; and experience teaches us more and more that in this cavity often lie the complications which keep up the suppuration in many obscure cases, or cause frequent relapses in cases apparently cured.

The complications found in the attic may be any or all of the first four mentioned, and it is by no means unusual for all four to be found in the same case. Those cases of tympanic suppuration in which we have a small perforation of the membrana flaccida only are particularly liable to develop complications in the attic, although we may find the same conditions with any tympanic suppuration, even when almost the entire drum-membrane has been destroyed.

The advantages to be gained by cleaning out all of the structures in the attic, the situation and the extent of the caries of the ossicles, and the influence of caries of these bones on the general suppuration of the tympanum, is the object of the present inquiry.

The diagnosis of caries of the ossicles can only be made with certainty by means of a bent probe which, touching the carious spot, gives to the finger the peculiar rough grating of carious bone; the disease may, however, be suspected in any case of long-continued suppuration in which the ossicles have not been already thrown off by the disease. The spots particularly to be explored are the external and medial surfaces of the head of the malleus, and of the body and long process of the incus; for, as is shown by my specimens, these are the spots most frequently affected.

I have records of sixty operations for the removal of carious ossicles, in which sixty hammers and fifty-two anvils were removed. They include all cases of this operation which I have done:

	Normal.	Carious.
Of the hammers:		
Head	24 (40 %)	36 (60 %)
Articulation	41 (68.3%)	19 (31.7%)
Neck	31 (51.7%)	29 (48.3%)
Short process	52 (86.7%)	8 (13.3%)
Manubrium	30 (50 %)	30 (50 %)
	178	122
Of the anvils:		
Body	8 (15.4%)	44 (84.6%)
Articulation	37 (71.1%)	15 (28.9%)
Long process	0	52 (100 %)
Short process	31 (59.6%)	21 (40.4%)
	76	132

Of sixty hammers seven were normal throughout (11.7%).

Of fifty-two anvils none were normal throughout (0%).

Of 300 anatomical portions of the hammers 178 (59.3%) were normal, and 122 (40.7%) were carious.

Of 208 anatomical portions of the anvil 76 (36.5%) were normal, and 132 (63.5%) were carious.

The most frequent spot of caries on the hammers was the head, which was carious in 60 per cent. of the bones; the next frequent was the manubrium, 50 per cent.; the next the neck, 48.3 per cent.; then the articulation in 31.7 per cent.: and the least frequently affected was the short process, in 13.3 per cent.

The most frequently affected spot on the anvil was the long process, which was carious in all the cases, 100 per cent.; next the body, carious in 84.6 per cent.; then the short process, carious in 40.4 per cent.; and the least frequently affected was the articulation, carious in 28.9 per cent.

In almost every case the anvil was more extensively diseased than the hammer.

Of the fifty-two anvils the long process was partially or wholly destroyed and disarticulated from the stapes in thirty-nine, or 75 per cent.

In every one of these cases persistent antiseptic cleansing

by the middle-ear syringe had been used for a long time, six weeks to six months, without any effect on the suppuration before the operation was resorted to. The cases, therefore, furnish, as far as their limited numbers will allow, a fair estimate of the value of the operation in itself, for the same treatment was carried out after as before the operation. Almost all of them were cases in which the suppuration had existed for months, and in many of them for from five to ten years.

All of the operations were carried out under ether narcosis, in the sitting position. With the exception of two or three of the earlier cases, any portion of the drum-membrane which existed was removed. I was early led to adopt this procedure, rather than to excise a small portion of the upper part of the membrane, because one of my very first cases developed a new cicatricial membrane, which closed the attic before suppuration had ceased. After dissecting off the drum-membrane as near its periphery as possible, and turning its posterior part forwards across the manubrium, with a Schwartze's tenetone the tendon of the tensor-tympani muscle is severed. Exploration with a fine probe will then usually tell whether the long process of the incus is still articulated with the stapes. If such proves to be the case, disarticulation is effected with an angular knife, and the long process of the incus pushed a little aside. The hammer is then seized with strong forceps, as high up as possible, — usually about the short process, — carried a little inwards to displace it from the attic, and then brought down and out. With a Ludewig's incus-hook, the incus is then swept backwards and downwards, till it appears in the meatus, when it is removed with forceps. The attic is then explored for desquamative products and granulations, which are removed by forceps, snare, curette, or Ludewig's hook.

Of the different steps of the operation, disarticulation of the incudo-stapedial joint is the most delicate and most difficult. Sometimes the joint can be seen; but often that portion of the operation must be done by feeling.

In only one instance — one of my earliest cases — have I met with an accident at this stage. In this case, the stapes was displaced and came away.

In seventy-five per cent. of my cases nature had already performed the disarticulation by a carious destruction of the end of the long process, so that the risks of this step were avoided.

In two cases, the operation was followed by facial paralysis, which lasted for three or four weeks, and then passed away entirely. Undoubtedly, the Fallopian canal, in its passage through the tympanum, was opened by the Ludewig's hook. The moral drawn from these two cases — which were among my earlier ones — was to keep the hook well toward the outer side of the attic of the tympanum; or, if it was necessary to pass the hook along the inner wall in order to bring the incus down, to use very little force. A twitching of the facial muscles, when the hook is in the neighborhood of the Fallopian canal, is a signal of danger, and should always be watched for.

The removal of the incus is not always easy. It is sometimes so firmly attached, by calcifications, that considerable force is necessary to displace it. In other cases, the attic is so narrow or irregular that it has to be worked down slowly. On the whole, I have found the Ludewig's hook a very satisfactory instrument; and I very much prefer it to any other instrument which I have yet seen. It is broad enough to sweep the attic effectually, strong enough to allow one to use sufficient force to displace an adherent bone, and its flat surface is very efficient in bringing down desquamative masses and granulations. In quite a number of the cases, a sweeping of the hook from in front backwards failed to engage and displace the anvil; but by reversing the process, and sweeping from behind forwards, the bone was brought down.

Bleeding can be greatly reduced by the use of cocaine. Adhesions of the drum-membrane to the labyrinthine wall require to be cut away; and, for this purpose, I have found Schwartze's tenetone very useful; for after cutting the tendon of the muscle, the adherent membrane can be readily dissected off, by carrying the knife downwards without withdrawing it.

The treatment after operation consisted in syringing with the middle-ear syringe, and the use of antiseptics applied

directly to the attic — most commonly, a saturated solution of boric acid, in absolute alcohol, for granulations; simply boric-acid powder for a slight serous discharge; and, latterly, a solution of carbolic acid, in pure glycerine (1–25), for simple suppuration.

The majority of cases were in hospital patients of the lower class, careless of personal cleanliness, and especially exposed, on this account, to a renewed infection of the necessarily exposed tympanum.

All of the cases were much less exposed to the risks of retained pus after the operation, even if the suppuration continued; for the clearing out of the attic and tympanum opens up these cavities in such a way that ordinary syringing, through the meatus, can cleanse every part. Before the operation this could not be done, even when the middle-ear syringe was passed directly into the attic. In this sense, every case would be said to be improved.

In regard to the suppuration itself, the results were various. In some, all suppuration ceased absolutely; the mucous membrane becoming dry, white, and hard — probably by a transformation of its epithelium into epidermis. In others, the discharge ceased entirely; but the tympanic membrane retained more or less of its mucous character, and was liable to secrete a minute quantity of mucus occasionally, under the irritation of a cold, or, perhaps, from dust, the discharge, in these cases, drying in situ, and forming a small crust. In others, the discharge diminished greatly in quantity after the operation, but did not cease while the patient was under observation. These cases I have called "improved." In some, the amount of suppuration was not diminished in the least. These I have called "not improved." In a considerable number of the earlier cases, the result is "unknown," from a failure of the records. Some ten cases, done within the last six weeks, are "too recent" to speak of the result. Some cases discharged apparently without suppuration; relapsed later, and became as bad as ever.

I have been able to examine twenty-six of the sixty cases one year or more after operation.

1. Eight were absolutely dry and had been so for one year or more.

2. Nine formed small crusts every few months.

3. Nine dry when discharged, relapsed with suppuration as abundant as ever. This gives seventeen or sixty-five per cent. successes, and nine or thirty-five per cent. failures. Of the remaining thirty-four cases:

4. Three were discharged dry, and nothing further is known.

5. Three were "improved," and nothing further is known.

6. Two were "not improved," and nothing further is known.

7. Sixteen are "unknown," and nothing further is known.

8. Ten are "too recent."

In percentages we have for the whole series of cases:

1st class . .	13.33%	6th class . .	3.33%
2d " . .	15.	7th " . .	26.6
3d " . .	15.	8th " . .	16.6
4th " . .	5.		
5th " . .	5.	Total . .	99.96%

Two cases originally discharged as "not improved," afterwards dried under home syringing, and when seen had been absolutely dry for one year. They are classed in the first variety. It would be reasonable to assume that some others of the "improved" and "not improved" varieties also dried eventually. Of the "unknown" and "too recent" cases, there is no reason why the same proportion of success should not be assumed as in the "known" cases.

In regard to the effects of the operation on the hearing, the records are not as complete as I could wish. In only eighteen of the cases have I been able to compare the tests one year or more after operation with the tests before operation.

Effects of the operation on

Hearing.	Discharge.				
	Dry.	Crusts.	Improved.	Not Improved.	Total.
Much improved.....	2	.....	.....	.....	2
Slightly improved.....	2	1	1	3	7
Same	1	2	.....	1	4
Worse	2	.....	1	2	5
Total	7	3	2	6	18

Of the five cases in which the hearing was worse after the operation, it may be said that in three of them the suppuration was not checked, and the falling off in hearing was gradual, and the result of the continued suppuration. Of the other two, in which the inflammation entirely subsided, one was the case already mentioned, in which the stapes was accidentally removed, and the other one of very offensive otorrhœa, through a small perforation of the membrana flaccida, but with very good hearing, and the loss of the focusing power of the drum-membrane and chain of ossicles diminished the hearing very perceptibly.

In most of the cases the direct effect of the operation upon the hearing was negative, for in 75 per cent. of them the incus had already been disarticulated by disease, and consequently any remnants of the conducting apparatus outside of the stapes were of no influence upon the hearing. The indirect effect was to free the cavity from obstructing pus, granulations, and desquamation, thus allowing the sound-waves to reach the stapes more directly and more fully.

In 52 of the 60 cases the anvil was found and removed. In every case the diagnosis of caries of the ossicles was confirmed.

In reviewing this series of cases I find myself somewhat surprised at the good effects of the operation in a large proportion of the cases, for it should be remembered that we

were dealing with the causes of suppuration in but a portion of the tympanum, and the same causes probably existed in other parts not reached by this method of treatment, to explain the failures. Granulations and caries are the two chief foci of inflammation in all chronic tympanic suppurations, and their existence in the additus, antrum, mastoid, or deeper portions of the temporal bone requires more than a clearing out of the attic and antiseptic treatment, with which alone I have dealt in this paper.

X.

A CASE OF CASTRATION FOR HYPERTROPHIED PROSTATE.

By M. F. Gavin, M.D.

This operation is still *sub judice*, and the history of this case recently operated upon may be of interest to the profession.

C. G., sixty-nine years old, a widower, entered Boston City Hospital, May 5, 1894, with the following history: Frequent and scalding micturition for five years, occasional pain over pubes, never any retention or dribbling. Examination by rectum showed a large, dense, symmetrically enlarged, smooth prostate, size of a large orange, occluding to considerable degree the rectum, the finger-tip reaching with difficulty the upper edge. The catheter was passed easily until the prostate was reached, which was surmounted with difficulty. The residual urine was ʒ v, was acid and contained pus, some bladder cells, a trace of albumen, and very few renal elements. In five days the patient was discharged with directions to return to hospital for catheterization weekly.

For four months patient followed this treatment, but the symptoms increased rapidly, and constantly recurring pain made more frequent catheterization necessary, and he was taught the use of the catheter with poor success, the prostatic urethra being too difficult for the patient to pass catheter through. He finally came to hospital with absolute retention which had existed for twenty-four hours with great pain, distressing tenesmus, coated tongue, tremulousness, and prostration.

A No. 9 Coudé catheter was passed with difficulty, and several ounces of cloudy urine withdrawn. Its s. p. was 1010, reaction and no albumen. Catheterization had to be

performed every four hours, day and night, because of the irritable condition of the bladder, which was quieted by morphia. Pain was still a prominent symptom, and the patient was unable to pass water at all. This condition continued for nearly three weeks, when the urine was still cloudy, acid, 1012, albumen a trace, and pus was found in the sediment. Castration was performed, and about eight hours after operation the patient had the first voluntary micturition for three weeks. Sixteen hours after the operation catheterization was needed. Voluntary micturition continued during the next day, and a second catheterization was demanded about forty hours after operation. From this time on no catheter was needed, morphia was omitted, and voluntary micturition, unattended by pain, continued. There was no more tenesmus or discomfort. At the end of a week the sutures were removed from the incisions, the wounds healing firmly. The prostate was then diminished markedly in size and consistency. The patient's nervous symptoms were quieter and disposition much more cheerful.

During the second week the patient was up and about the ward in clothes, passing his water during the day about every three or four hours, and three or four times a night. The force of the stream was increased. The character of the urine was unchanged, except that there was a little less pus. The urine chart then showed a polyuricas as high as ʒ 120 being passed in twenty-four hours, the average being about ʒ 98. Three weeks after the operation the prostate was diminished one-half, and in five weeks was only one-fifth of the size before operation. The patient was then discharged. The urine was then ʒ 70 in twenty-four hours, acid, albumen a trace, pus much less in quantity, residual urine ʒ iii.

The patient reports from time to time at the hospital. He is grateful and enthusiastic, free from pain or discomfort, passes water every four hours during the day and two or three times at night. The residual amount is ʒ ii–iii and is clear, s. p. 1010–14, trace albumen. Patient has gained thirty pounds since leaving the hospital. Prostate is now about one-sixth size before operation.

This case has considerable interest in many ways; it goes

to show that permanent benefit does follow the operation of castration for hypertrophy of the prostate. When contrasted with the many other operations that have been suggested, it has much to commend its use. It is rather repulsive to the average man to think of the loss of his testicles, but when we consider that this operation is only called for late in life, when the activity of those organs, to say the least, is on the wane, it must lose much of the repulsiveness that has been urged against this operation. The operation is a simple one and does not entail much risk to life. Of course it is only for a certain class of cases that this operation is called for, — persons unable to follow out systematic course of palliative treatment; where the passage of a catheter is attended with the loss of blood or great pain; where obliged to work and exposed to the changes in the weather, and where the liquor habit prevails.

The subject of this operation had tried the palliative treatment faithfully without any gain; in fact, he was losing flesh and strength. He was told of the different operations, — perineal drainage, supera-pubis, cystotomy, prostatectomy, and castration, — but was willing to allow us to use our better judgment. When we think of the suffering that follows hypertrophy of the prostate, how difficult to reach even to afford relief, living with a disorganized condition of the bladder and later the kidneys, any remedy ought to be gladly welcomed by the surgeon. I have been greatly impressed with this case and the result that followed operation.

The suffering of a woman in advanced ovarian disease is no greater than that of the man with an enlarged prostate and its consequences, and if ovariotomy is done to relieve the one, why not castration for the latter?

XI.

AN ANALYSIS OF ONE HUNDRED CASES OF URETHRAL STRICTURE OPERATED UPON BY VARIOUS METHODS.

By Francis S. Watson, M.D.

This series comprises all the hospital cases operated upon by the author in the last eight years, with the exception of those treated by gradual dilatation.

The operative methods were as follows:

Internal urethrotomy for strictures anterior to five inches:	
(*a*) By Otis's method	37 cases.
(*b*) By Maisonneuve's method	13 "
Total	50 "
Internal urethrotomy for strictures of the membranous urethra	2 "
Combined internal and external perineal urethrotomy	24 "
External perineal urethrotomy alone	13 "
Divulsion for strictures of the membranous urethra	4 "
Internal and external urethrotomy (by Gavin's method)	5 "
Resection and suture for anterior stricture	1 case.
Supra-pubic cystotomy and subsequent external perineal urethrotomy	1 "
Total	100 cases.

The total mortality was four per cent.

One death followed Otis's internal urethrotomy for a stricture of the anterior urethra of twenty-four calibre in a

young man of twenty-eight years of age, and was due to a typical attack of urinary fever and partial suppression of urine. Death occurred ten days after the operation.

One death followed internal urethrotomy for a tight stricture of the membranous urethra in a patient of forty-eight years of age, and was due to urinary fever and septic infection from the wound.

Two deaths followed external perineal urethrotomy alone. Both patients were over seventy years of age, both had extensive urinary extravasation, and both were in collapse when they came to operation.

Maisonneuve's method was employed in all cases of anterior stricture whose calibre was less than fifteen of the French scale, which is the smallest number that will admit an Otis instrument. These operations were completed with the Otis instrument, by cutting to the full calibre of the urethra.

There was urinary extravasation, previous to operation, in four of the cases in which external perineal urethrotomy alone was performed.

External perineal urethrotomy was performed in forty-three cases altogether. Of this number six were done without a guide; in twenty-six, Wheelhouse's method was employed, a guide being passed after laying open the urethra on the staff. In five cases the perineal operation was done in combination with an anterior internal urethrotomy, stricture of the anterior urethra alone being present, with the object of preventing the passage of urine over the wounds of the latter, in accordance with the practice recommended by Reginald Harrison, his belief being that urinary fever is due to the contact of urine with the freshly cut surfaces. He claims almost entire immunity from urinary fever since adopting this practice. In the five cases above noted, such immunity was not secured, for two of the patients suffered moderately severe attacks of urinary fever after the operation. There were five cases treated by a method recently suggested to me by my colleague, Dr. Gavin, which its author permits me to describe.

This method is applicable to all cases of tight or resilient stricture of the bulbous or membranous urethra in which it

has been decided to perform external perineal urethrotomy, and which will admit a filiform guide. Such cases may, of course, be dealt with by internal urethrotomy, but the best surgical opinion favors the external operation as being safer in this condition, because better drainage is secured, and so there is less danger of septic infection, and because hemorrhage is more easily controlled. The usual manner of performing the external operation is to open the urethra upon a grooved staff just above the stricture, and then to divide it with a knife, following a filiform guide which has been passed through it. Dr. Gavin's modification consists in performing first an internal urethrotomy with a Maisonneuve instrument, thus dividing all the anterior strictures, if present, as well as the posterior ones, with one clean cut of the knife. This allows the passage of a large grooved staff, upon which the external perineal cut can now be made with one movement, thus completing the whole operation in two cuts. It has the advantage of rapidity, and of making certain the complete and clean division of all strictures with one cut of the knife.

The last case of the forty-three was treated by supra-pubic cystotomy at first, external perineal urethrotomy being done subsequently. This case was as follows: N. S., aged twenty-six, came to the hospital suffering from retention of urine, due to an acute gonorrhœa in addition to a tight stricture of the membranous urethra. It was found impossible to pass any catheter into the bladder; the latter was greatly distended, and the patient was suffering. Owing to the danger of infecting the wound with the gonorrhœal pus, it was thought unwise to operate upon the stricture at that time, and the urinary retention was relieved by performing a supra-pubic cystotomy. In doing this the fold of the peritoneum was accidentally knicked; this wound was united with two silk sutures and gave no trouble afterward; double drainage tubes were worn for nearly one month; the wound was then allowed to close; meanwhile the gonorrhœa had been successfully treated, and the patient was ready for the second operation, which was done by Dr. H. W. Cushing, the stricture being divided by an external perineal operation, which resulted successfully. The patient was seen six

months later and reported that he was entirely free from all symptoms. The cystotomy wounds healed in about ten days after the removal of the drainage tubes.

The single case in which resection and suture were employed was that of a young man with a traumatic stricture of the anterior urethra and resulting urethral fistula; about quarter of an inch of the urethra was resected, and the divided ends of the urethra were united with eight fine silk sutures; first intention of the wound followed, but a stitch abscess reopened it for a few days; three weeks after the operation, the urethra admitted easily a No. 28 sound, final healing having taken place.

When practicable the patients were kept for two days in the hospital before operation, and during this time were given from twelve to twenty grains of boracic acid by mouth daily, to sterilize the urine, as far as possible. Just before operation the urethra is washed out with a four per cent. solution of boracic acid, and afterward both urethra and bladder are thoroughly irrigated with the same solution; this has been the routine practice during the last four years, and since its adoption constitutional disturbance following the operations has been much less frequent. The administration of aconite, quinine, or morphia after the operations has not in the author's experience prevented urinary fever, though the latter (morphia) as often seemed to shorten the duration of the chills. The most efficient prophylaxis consists in securing the most thorough asepsis possible of the field of operation.

Of the whole number of cases operated on there was no elevation of temperature or any constitutional disturbance whatever in sixty per cent. This percentage of *immunity* was distributed as follows with respect to the methods of operation:

Internal urethrotomy . . .	34	cases	out of	52
Combined internal and external urethrotomy	18	"	"	29
External urethrotomy . . .	6	"	"	13
Divulsion	2	"	"	4
Total	60	"	"	98

A catheter was *not tied* into the bladder after internal urethrotomy or divulsion in any case. A catheter *was tied* into the bladder in all but eight cases of external perineal urethrotomy. Of these eight, four had one or more chills and rise of temperature, a very large percentage as compared with the cases in which a catheter was tied in; in the four in which constitutional disturbance occurred it ceased on introducing a catheter. This experience suggests that the catheter is in some measure at least a prophylactic measure against urinary fever in cases of perineal section. A larger experience in the same direction has confirmed the author in this view, whereas it has not been so with regard to internal urethrotomy.

In eighty per cent. of the forty cases in which constitutional disturbance followed the operations, the chill or rise in temperature occurred with or immediately after the first urination or the first passage of sounds. To guard against this so far as possible, the urethra is thoroughly irrigated with a four per cent. solution of boracic acid, just before and just after the operation, the first urination, and first sound-passing; the instruments are warmed, used with great gentleness, and must be absolutely clean. In cases of very dense or tortuous strictures of the deep urethra which were treated by external perineal urethrotomy, a catheter was left in through the whole urethra for a much longer time than usual, sometimes for ten days, — this in order to allow the divided portion of the urethra to heal around the catheter. A soft red rubber catheter is the one always selected for this purpose. After the removal of the catheter in cases of external perineal urethrotomy, it is the routine practice to pass sounds of the size to which the stricture has been cut, every second day, until healing is complete; this is also done after internal urethrotomy. In the latter, absence of blood on withdrawal of the sound is taken as a sign that healing has occurred; this generally takes place in about a fortnight. The shortest time in which healing took place after the external operation was in forty-eight hours, in one case: in four others it occurred in four days; no catheter was used in any of these cases; the longest time before healing took place in any case was six weeks; the average length of time was twelve days.

There were two cases in which urinary fistulæ resulted. In the cases of internal urethrotomy alone, it is the custom to keep the patients in bed until the fifth day, whether they have any symptoms or not; that gives them twenty-four hours' quiet after the second passage of sounds. If there are no symptoms then, they are discharged from the hospital, and are directed to the Out-Patient Department for the three or four final sound-passings, after which they should come for examination once in six months.

In the four cases of divulsion, Bigelow's instrument was used once, Voillemier's twice, and once the stricture was ruptured by the passage of steel sounds of increasing sizes. Two of the patients had attacks of urinary fever, in none was the catheter retained after operation; the two patients who did not have urinary fever left the hospital well at the end of a week; the other two were well at the end of a fortnight and three weeks respectively.

There were four cases of traumatic stricture altogether. Three were situated in the perineal, one in the anterior urethra.

The highest temperature noted after operation, in any case, was 105°. This was after an internal urethrotomy. The patient made a good recovery, however.

Accidents occurring in connection with operations:

In one case of internal urethrotomy for anterior stricture, there was urinary infiltration; and in one case of external perineal urethrotomy, there was a severe secondary hemorrhage. Neither accident led to serious consequences.

Acute urethritis followed internal urethrotomy in four cases.

Final Results.

Internal Urethrotomy.

Operation eight years ago, in five cases; examined within the past year, two. No recontraction in either case. The patients are free from symptoms of stricture.

Operation seven years ago, in four cases; examined within the past year, one. No recontraction.

Operation six years ago, in ten cases; examined within the past year, two. Recontraction in one, at the site of the original stricture.

Operation five years ago, in six cases; examined in the last year, two. No recontraction in either case.

Operation four years ago, in eight cases; examined in the last year, four. Recontraction in one.

Operation three years ago, in four; examined in the last year, one. No recontraction.

Operation two years ago, in eight; examined within the last year, four. No recontraction in any of them.

Operation one year ago, in five cases; examined in the last year, five. No recontraction in any of them.

This makes a total of fifty cases, in which internal urethrotomy alone was done. Twenty-one of these have been examined within the past year. Of this number two have recontracted, — one within six, and one within four years after the operation, — or about ten per cent. of the cases re-examined. Two of the four cases of divulsion have recontracted, one within a year, and one within eighteen months. But five cases of the external operation have been seen since the operation. There was recontraction in two of these within three years.

An estimate of the final results of operations with hospital patients is necessarily unsatisfactory, owing to their being lost sight of, as a rule. Twenty per cent. of this series, which reported for subsequent examination, is an unusually large number to have done so. But even from this number no inference as to the permanency of the results of operation can be drawn from those cases operated upon within the last three years; nor can a just estimate of the frequency of permanent results be formed unless all the cases have been re-examined. The partial estimate given above, therefore, is all that the author has attempted. It may be fairly assumed that this class of patients furnishes the least favorable results, both as to mortality and later conditions; which compare — especially the latter — unfavorably with the author's experience in private practice.

In two of the cases reëxamined, — which had been subjected to internal urethrotomy, one at six and one at seven years, previously, — endoscopic examination showed admirably the condition of the cicatrix claimed by Otis to be formed after his operation; namely, a thin, pliant, elliptical

scar, showing no tendency to contraction; the urethra being, on the contrary, wider at the point corresponding to the middle of it than anywhere else. The author has been especially struck with the increased facility in performing the external perineal operation afforded by the modification described as that of Dr. Gavin, and would call attention to its technique.

The mortality of this series corresponds with that ordinarily given by various authors, in estimating it from large numbers of cases. The writer, for example, found the mortality to be a little more than two per cent. in a series of six thousand cases of internal urethrotomy, performed by various operators; and the same in a large number of cases of divulsion. In external perineal urethrotomy, it was about six per cent.

XII.

NOTES ON PELVIC INFLAMMATION.

By Charles M. Green, M.D.

About twenty-five per cent. of the patients entering the service for diseases of women are suffering with some form of pelvic inflammation. This proportion is considerably less than the ratio of pelvic inflammation to all other classes of disease peculiar to women, as observed in the community at large; for the reason that the milder cases can be cared for at home, or in out-patient departments, and do not seek admission; and because, also, the large number of cases sent in for plastic operations, for the removal of new growths, or for puerperal convalescence precludes the possibility of admitting many chronic cases of pelvic inflammatory trouble.

Under the general heading of pelvic inflammation are included the various forms of salpingitis, ovarian abscess, pelvic abscess, and those conditions variously designated under the terms of perimetritis, parametritis, pelvic cellulitis, and pelvic peritonitis; vaginitis and uncomplicated endometritis are excluded. This classification is adopted in accordance with the present views of pelvic pathology and ætiology. It is now generally accepted that the various stages of pelvic inflammation are but differing degrees of one and the same morbid process, of which the cause is to be found, in the vast majority of cases, in some form of septic or specific infection. Whether the pathogenic micrococcus effects an entrance by impure sexual contact, or by septic instruments or examining fingers, a flame is lighted which may be limited to the Fallopian tube, or which may extend through either the fimbriated extremity or the tube-wall, and sweep through the pelvis, involving the ovary, the pelvic peritoneum and cellular tissue, resulting, perhaps, in ab-

scess formation, which may destroy not only the uterine appendages, but the life of the patient as well. The degree to which any pelvic inflammation may attain of course depends chiefly on the nature and virulence of the infecting virus, and is also influenced, to some extent at least, by the environment of the patient, and by the promptness and intelligence of the treatment.

It is not intended in this brief paper to deal with statistics. In no class of cases, so far certainly as hospital practice is concerned, are statistics of less value than in pelvic inflammations. The stay in hospital is necessarily limited by personal and prudential reasons; and even though it were possible to keep patients until all evidence of disease has disappeared, most patients desire discharge as soon as subjective symptoms have vanished, even though examination still reveals the presence of exudate or other physical signs of disease. The ultimate results of treatment are generally, therefore, unknown. If, in a given case, the indications point to cœliotomy, and if the patient submits to the removal of diseased organs, it may be possible to discharge the case well; but in the relatively large proportion of cases in which abdominal section is not called for, or is declined, and in which less radical measures succeed in removing pain, tenderness, hæmorrhage, purulent discharges, or collections of pus, the patient can be discharged only as symptomatically well or relieved. There is good reason to believe that large numbers of these cases remain symptomatically well; that old exudates become organized; and that uterine appendages, even although functionally useless, give no further trouble. On the other hand, it is well known that in such cases recurrent attacks are not infrequent, and that the patient must sometimes ultimately choose between surgical interference and years of quasi invalidism. Certain it is that statistics of pelvic inflammations are of slight value unless based on a considerable number of cases observed through a period of years.

Most cases of pelvic inflammation can be classified under one of the following five heads:

I. *Salpingitis.* — One or both tubes may be affected, and there is an accompanying endometritis. There is a copious

catarrhal discharge from the tube, in which case there is no distention, and the tube usually cannot be palpated, the diagnosis of catarrhal salpingitis being made in such cases from the history and symptoms rather than from physical signs. Or the proximal end of the tube may be occluded, and distention thus result. In these cases there is no interstitial infiltration of the tube, and no pelvic exudation. When there is free drainage from the tube, pain is not a prominent symptom, and such cases doubtless often result in spontaneous cure. But when there is proximal occlusion of the tube and hydrosalpinx results, pain is sometimes severe. Hæmorrhage may or may not be a symptom. In most cases there is a slight elevation of temperature.

The treatment adopted for this class of cases is as follows: The patient is kept in bed, the bowels are freely opened, and such general measures taken as may be indicated in each case. Indeed it may be said that few cases of gynæcological disease are uncomplicated with some form of general functional disturbance, and general medical treatment, therefore, forms an important part in the management of all classes of pelvic inflammation. After the vagina has been disinfected with sublimate douching and scrubbing, the uterus is dilated and thoroughly curetted, washed or swabbed out, and packed with iodoform gauze. The vagina is also lightly filled with the same material. After five or six days the vaginal gauze is removed and the vagina cleansed. In some cases the intra-uterine gauze is found to have been expelled, in others it has remained *in situ*. It is usually best to renew the gauze at this time, and keep the uterus packed for ten days, after which time all dressings are removed, and hot douches are systematically given. In some cases all discharges will have ceased in two or three weeks; but in others a few intra-uterine applications of iodine are necessary. There is no present known method of treating the tubal lining directly; but by the above measures the uterus is freed from pathogenic bacteria, and free drainage of the tube facilitated. In cases of occlusion of the tube and a consequent hydrosalpinx, whether by removal of the occluding tissue with the curette or by the subsidence of swollen endothelium, it is often found that the tube discharges itself and

the tumor disappears. Or the tumor may slowly subside by absorption and inspissation of its contents. Cœliotomy is rarely indicated. The above treatment is by no means invariably successful, for in some cases the offending micrococcus has penetrated the wall of the tube, or passed from the distal end, and we have ultimately to deal with an interstitial salpingitis, ovaritis, peritonitis, with perhaps abscess formation.

II. *Tubo-Ovarian Inflammation.* — Under this conventional head are classified cases of interstitial salpingitis and ovaritis, of pyosalpinx and ovarian abscess. There is always more or less peritonitis in these cases, especially in the acute stage, and usually some cellular infiltration; the amount of exudate is not usually sufficient, however, to prevent diagnosis, provided the patient is placed under ether. The uterus is usually more or less fixed, although in some acute cases this organ is fairly movable. There is usually considerable pain, the amount depending on the extent of the peritoneal involvement, and exquisite tenderness on palpation. The elevation of temperature is, of course, most marked when there is considerable pus formation.

In the treatment of this class of cases, the advisability of cœliotomy is not at first considered, except in well-marked and unmistakable cases of ovarian abscess, or pus-tube of considerable size. In such cases palliative measures have no part, and immediate abdominal section is advised. Except in these cases, however, conservative and palliative measures are first given a full and patient trial. It is first sought to cut short the peritoneal inflammation and thereby relieve the pain, and then to promote the absorption of exudate, and thus reveal more exactly the size and condition of the uterine appendages. It is often surprising to see what changes will follow the proper use of the hot douche, the free application of glycerine, and perhaps of ichthyol, combined with absolute rest in bed and attention to the eliminative functions. In some cases it is thought best to curette the uterus, to remove a lingering endometritis and lighten the somewhat enlarged organ. If there is no suppuration in the ovary, and no large purulent accumulation in the tube, conservative treatment is, in a very large proportion of cases, successful. That is to say, the symptoms are relieved, the

general health is restored, and the only evidence of disease is to be found in a somewhat thickened, and perhaps adherent, tube. In some cases, although probably rarely, the tube discharges its contents through the uterus; but in others the tubal contents become inspissated and cheesy, and the tube ceases to give trouble, although functionally useless. Of course such cases may have recurrent attacks, and cœliotomy may ultimately have to be performed; but undoubtedly many cases remain symptomatically well.

III. *Pelvic Inflammation, with Exudation preventing Accurate Diagnosis.* — Under this head are classed those cases in which the exudation far exceeds that found in the last preceding group, and in which the inflammation involves the entire pelvic peritoneum, the plastic lymph not only filling the pelvis, but pushing up or surrounding the lower coils of intestine, so that a distinct tumor is felt considerably above the pelvic brim. The pelvic cellular tissue is infiltrated, the uterus is fixed, the vaginal vault is hard and board-like, and the uterine appendages are not differentiated. Such cases enter the hospital in the acute stage, with a high temperature, vomiting, great pain, and marked prostration. Except in the less common puerperal cases, in which infection may have found entrance along the lymphatics, or except again in the still less common cases in which a diseased appendix has been the prime cause, this form of pelvic inflammation must have originated in an endometritis and a salpingitis, although in the development of the case the smaller, kindling flame is swallowed up in the pelvic conflagration.

In the treatment of these cases, in the acute stage, the application of ice-bags to the hypogastrium has been found of great value, the pain being apparently thereby diminished, and the shrinkage of the tumor begun. In the few cases in which ice has not been well borne, poultices have been used. Morphia is given as sparingly as possible, but is generally necessary in the beginning. For a few days saline cathartics are freely used, to the extent of effecting five or six dejections daily, after which the bowels are simply kept soluble. Under this treatment the writer has seen the most happy results — the entire disappearance of pain, the subsidence of

fever, the absorption of exudate, a complete or partial mobility of the uterus. It has generally been impossible to keep patients who have reached this stage of recovery, as they wish to return to their work. A small amount of hard, organized exudate is usually to be felt behind the uterus, which may or may not ultimately disappear, but which, at all events, no longer causes symptoms. In some cases the absorption of exudate is hastened by the use of hot douching and glycerine, and the application of iodine. All cases do not follow this highly favorable course. In some, after the partial absorption of the exudate, diseased appendages are clearly made out, and the case is then reclassified under the preceding head, to be treated surgically or with palliative measures, according to the indications. In others, more or less extensive suppuration takes place, and the case passes to the next classification, that of pelvic abscess.

IV. *Pelvic Abscess.* — Under this head are grouped those cases with more or less extensive exudation, sufficient to mask the appendages, in which suppuration is no longer confined to the tube and ovary, but has invaded the plastic lymph. There may be one large pus-sac, or several smaller ones which may or may not subsequently communicate. There is considerable pain, prostration, and elevation of temperature; fluctuation is evident by abdomino-vaginal or abdomino-rectal palpation. Sometimes the tumor lies low in the pelvis, with bulging of the pus-sac into the rectum and vagina; sometimes the lower border of the tumor is not within easy reach, or, at least, shows no signs of pointing on its vaginal or rectal aspect. In the former class of cases, the tumor is aspirated, then opened, and the pus evacuated, the sac washed out with a weak sublimate solution, and drained with a T rubber tube. Daily washing is then practised through the tube until the cavity shrinks and all discharge ceases. Under this treatment most cases do well and symptomatically recover, although usually some exudate remains at the time the patient leaves the hospital.

In the second class of cases, when the tumor lies high in the pelvis, it is not considered good surgery to approach it from below. Sometimes pointing is found close under the abdominal wall, and the abscess may be opened by a small

abdominal incision, washed out and drained as before. But in most cases, it is desirable to perform cœliotomy, with a view not only to evacuating pus, but to removing diseased, and perhaps disorganized, appendages.

V. *Peritonitis, with Retro-Displacement of the Uterus.* — It is not intended to classify under this head cases of tubo-ovarian disease, or indeed any of the forms of pelvic inflammation previously mentioned, which happen to be complicated with retro-displacement of the uterus. There is a not inconsiderable number of cases, in which there is a sub-acute peritonitis associated with adherent retroversion or flexion in which there is no discernible disease of the appendages. In some cases, examination under ether reveals diseased appendages, prolapsed and adherent, behind the uterus; but these cases are classified elsewhere. The patient enters with moderate fever, considerable pain in the back and pelvic distress, marked tenderness on vaginal palpation, more or less uterine discharge, and often a history of dysmenorrhœa. There is usually an endometritis and considerable impairment of the general health. Often there is a history of an injudicious use of pessaries, in the hands of physicians who recognized the displacement, but not the accompanying peritonitis. There is also a history of long semi-invalidism, or of periods of comparative health interrupted by exacerbations of the chronic inflammatory trouble. The nervous system is often seriously affected.

In these cases, general medical treatment for the relief of nervous debility, dyspepsia, and other functional disturbances, is strongly indicated. The local treatment is first directed to the removal of pelvic pain and tenderness. Wool tampons, well charged with glycerine, or with a ten per cent. mixture of iodoform and glycerine, have proved efficacious; ichthyol, also, has stood the test of experience. As soon as the pain and tenderness are somewhat abated, it has been found beneficial to curette and disinfect the uterus, and pack it with gauze. This procedure lightens the organ, removes endometritis, and checks the catarrhal discharge. Pain and tenderness having been allayed, the patient is then able to submit to carefully adjusted mechanical pressure, to overcome the retro-uterine adhesions. In some cases, when

the patient can be kept sufficiently long, the uterus can be completely replaced, and held with a suitable pessary; or the uterus can be supported in an improved position with an elastic, soft-rubber pessary, which can be worn with comfort and without danger. It is a noticeable fact, however, that many cases feel entirely well on the subsidence of peritonitis and after the restoration of general health, the retro-displacement *per se* causing no symptoms.

XIII.

FRACTURE OF THE HUMERUS FROM AN UNUSUAL CAUSE.

By G. H. Monks, M.D.

A well-built and apparently healthy man, aged twenty-seven, entered the Boston City Hospital August 8, 1894, having sustained a transverse fracture of the right humerus, just below the insertion of the deltoid. The patient had first been seen by Dr. John Gillespie, of Roxbury, who recommended him for admission to the hospital.

The following is the history of the injury. The patient, C. C., and a friend of his were testing their strength of arm by that well-known method of grasping right hands, palm to palm, over a table, and while the elbows rest upon the table each tries to push his opponent's hand down. I do not know of any English name for this sport, but Hamilton says that the French call it "tourner poignet," or the game of turning wrists. The illustration shows the position taken by the contestants in this sport. The elbows should, however, perhaps be a little closer together than is shown in the

photograph, so that the forearms may be more nearly parallel.

C. C. and his opponent were both exerting considerable force, and for a time neither seemed to gain any advantage, until, finally, C. C. began to push his adversary's hand down. At this moment the adversary made a sudden effort to regain his position, using all his force, when with a snap, so loud that it could be heard all over the room, C. C.'s humerus broke, and his arm fell helpless upon the table.

As to the subsequent history of the case there is nothing noteworthy. The arm was immobilized in the usual fashion in coaptation splints, shoulder-cap, etc., and in the course of six weeks the fragments were firmly united without deformity, and soon after this the arm appeared to be as strong and useful as before the accident.

Several cases of fracture of the humerus during the carrying out of the above form of sport have been recorded, but so few are they as to justify the publication of each new case. Hamilton states that he has seen one case of the kind, and refers to six or seven others which he has found recorded. Stimson ("Treatise on Fractures") mentions the fact that Gurlt gives eleven cases of the sort. It is probable that some of Gurlt's cases are included among those mentioned by Hamilton.

The mechanism of a fracture of the humerus from the above cause is interesting. Apparently the break is occasioned mainly by a twist, or torsion of the shaft of the bone. While the muscles of the upper end of the bone are attempting to rotate the bone inward, the adversary's force (transmitted through the bones of the forearm to the lower end of the humerus) tends to rotate it outwards. The upper end of the bone is therefore forcibly rotated inwards and the lower end is rotated outwards. It is thus evident that when these two opposing forces are great enough, and the other parts do not give way, the humerus must break by extreme torsion somewhere between the two extremities. This must be especially true when the fracture occurs while the forearm is flexed at a right angle on the upper arm. When, however, the shoulder is brought much nearer to the hand, as is often the case during the greatest efforts, and a fracture

occurs, another element probably comes somewhat into play, and the bone breaks by a combination of torsion and outward bowing. Torsion still remains, however, the principal mechanical factor in causing the break.

While the etiology of this fracture is undoubtedly muscular action, it seems to be a rather unusual form of it, and the break might be described as one due to torsion of the shaft of the humerus, caused by prolonged muscular action against resistance suddenly and unexpectedly increased.

In closing, I wish to say that careful investigation was made respecting the existence of syphilis in this case, with negative result. There was also no evidence of the presence of fragilitas ossium.

XIV.

DIAGRAMS OF ELBOW LANDMARKS.

By H. L. Smith, M.D.

It is sometimes useful, especially for records, to have some means of graphic representation by which normal and abnormal outlines of the elbow may be preserved and compared. A method of taking such tracings is represented in figure 1. After making a mark vertically beneath each of the three prominences, it is only necessary to indicate the general direction of the arm. Figure 2 shows the outline thus obtained. It would not be difficult, probably, to devise apparatus which would give more exact results, but, in practice, the simple method here used has proved sufficiently accurate, while it has the great advantage of being applicable to all cases, and under all conditions.

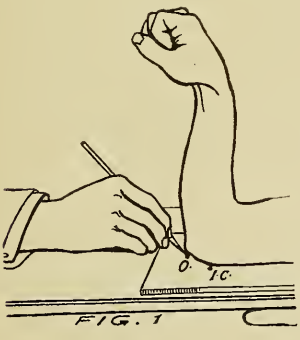


FIG. 1

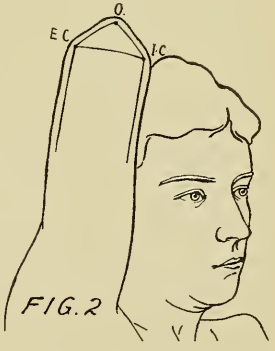
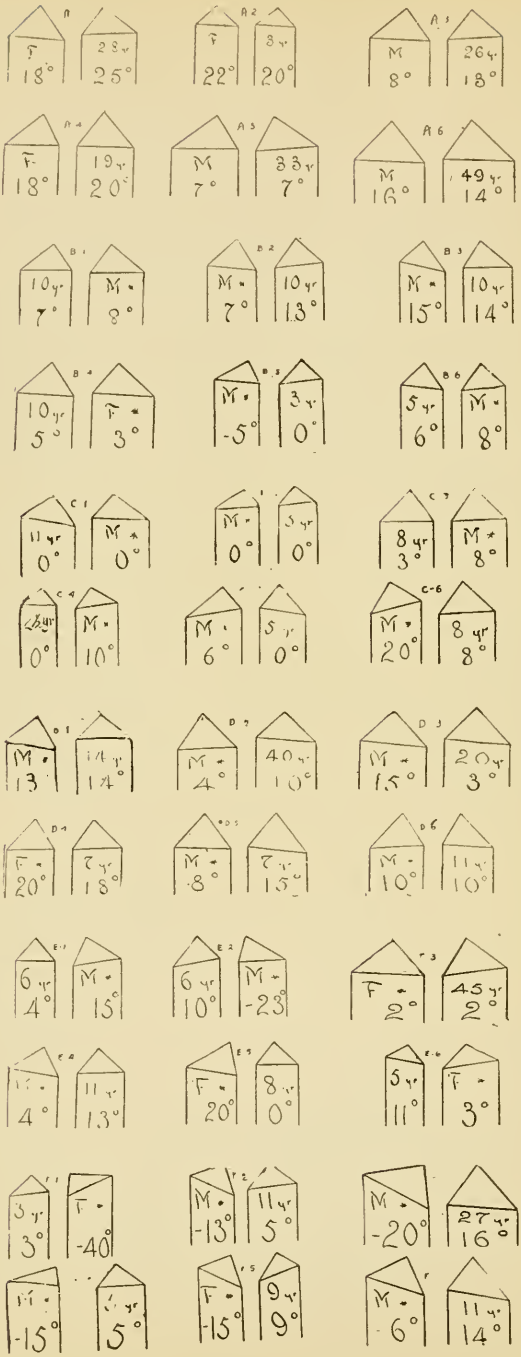


FIG. 2

In uninjured arms, the "elbow triangles" thus formed appear to be essentially similar figures, the angle at the olecranon being usually about 87°.

The points to be noted in studying the figures, are: (*a*) the distance between the condyles; (*b*) the direction of the


A1
F 18° 28 yr 25°
A2
F 22° 8 yr 20°
A3
M 8° 26 yr 18°
A4
F 18° 19 yr 20°
A5
M 7° 33 yr 7°
A6
M 16° 49 yr 14°
B1
10 yr 7° M 8°
B2
M 7° 10 yr 13°
B3
M 15° 10 yr 14°
B4
10 yr 5° F 3°
B5
M -5° 3 yr 0°
B6
5 yr 6° M 8°
C1
11 yr 0° M 0°
C2
M 0° 5 yr 0°
C3
8 yr 3° M 8°
C4
M 10°
M 6° 5 yr 0°
C-6
M 20° 8 yr 8°
D1
M 13° 14 yr 14°
D2
M 4° 40 yr 10°
D3
M 15° 20 yr 3°
D4
F 20° 7 yr 18°
D5
M -8° 7 yr 15°
D6
M 10° 11 yr 10°
E1
6 yr 4° M 15°
E2
6 yr 10° M -23°
E3
F 2° 45 yr 2°
E4
11 yr 4° 11 yr 13°
E5
F 20° 8 yr 0°
E6
5 yr 11° F 3°
F1
3 yr 3° F -40°
F2
M -13° 11 yr 5°
M -20° 27 yr 16°
M -15° 5°
F5
F -15° 9 yr 9°
F
M -6° 11 yr 14°

line joining the condyles (not identical with the joint-axis), as compared with the axis of the arm; (*c*) the position of olecranon compared with the condyles and with the axis of the arm; (*d*) the general shape of the triangle.

The diagrams here given are reduced copies of actual cases. There are six examples of each variety, save of the T fractures, E5 and E6 being introduced to show how the diagrams above may sometimes aid in diagnosis. The pairs represent the left and right arms, the former always first. In each case the sex and age are given, and the injured arm marked with a star. The figures indicate the amount of the "carrying" angle, the minus sign showing that it is reversed, thus causing the "gun-stock deformity." The series are as follows: A, normal arms; B, dislocations; C, external condyle; D, internal condyle; E, 1, 2 : 3 and 4, T fractures; 5 and 6 called external condyle, but former (as seems likely also, on other grounds) probably T or transverse, the latter very likely of external condyle, as reported; F, transverse fractures.

Dislocations vary little from the normal. Fractures of either condyle alone show less difference than might be supposed. The greatest change is seen after T fractures and transverse fractures. It is after these injuries, as will be seen from a study of the angles given, that the normal humero-ulnar or "carrying" angle becomes reversed, forming the so-called "gun-stock deformity," or cubitus varus.

It is impossible, in the space at command, to give a thorough analysis of these drawings, or to more than hint at their possible utility. For example, diagrams made before and after the reduction of a fracture are sometimes very striking.

Although no diagrams have been introduced as evidence, it may be noted that another series of tracings may be had by placing the forearm on the table, with the arm vertical, the resulting outline showing any forward or backward displacement of either condyle.

XV.

THE FLAXSEED-MEAL POULTICE AS A STERILE DRESSING.

By R. W. Lovett, M.D.

For an indefinitely long time the flaxseed-meal poultice held its place as an invaluable surgical dressing. With the advent of antiseptic surgery, however, it began to be condemned as dirty; and its use has been generally discountenanced by surgical writers, in spite of the fact of its undoubted practical utility in many conditions. The present paper represents an attempt to determine by experiment the question, whether this form of dressing can be considered a sterile one.

As an instance of the practical usefulness of flaxseed-meal poultices the following series of cases may be mentioned. After operations for chronic or acute glandular abscess, the writer has treated cases in two ways. In the first series of cases a wet corrosive dressing, with perhaps iodoform, was at once applied, and so far as possible was kept continuously wet. In the second series of cases a flaxseed-meal poultice was used from the first, and was changed every two or three hours. The cases observed have amounted to some forty in number altogether; and in those treated by the flaxseed poultices the patients suffered less pain, the induration disappeared more quickly, and the progress was in every way more favorable than with the corrosive dressing. To a certain extent the same is true with regard to felons and similar inflammatory conditions.

The following experiments deal with the flaxseed-meal poultice as a dressing considered from a bacteriological standpoint:

(1) In the diphtheria ward some flaxseed-meal was taken from a tin box in which it was kept, and boiling

water was poured upon it in the ordinary copper poultice-dish. It was kept hot over the gas heater for three minutes, a little hot water being added from time to time. It was then poured out in the usual way, smoothed with a clean knife, and covered with a piece of gauze not sterilized. A culture from the inner surface of the poultice, including the gauze, showed it to be absolutely sterile.

(2) In the surgical out-patient dressing-room, where fifty to one hundred wounds are dressed daily. The air in this room is foul on account of the large number of patients always there, and on account of the presence of many foul leg-ulcers, etc. A poultice was made in a clean copper tin by pouring boiling water upon the meal. The meal was taken from a tin box kept in the room. The water and meal were mixed with a knife. No precautions were taken to render the poultice sterile. A culture from the meal as it was poured out was sterile.

(3) Ordinary gauze, not sterilized, was placed over the inner surface of the meal. A culture showed a few cocci resembling pus cocci and a few thin bacilli.

(4) The hands were sterilized; the poultice dish was thoroughly cleansed; the meal and water were heated for three minutes over the heater.

Culture from the meal as poured out showed a few long, thick bacilli, probably non-pathogenic.

(5) Sterilized gauze was placed over the inner surface of the meal and wet in the moisture of the poultice. A culture from this was sterile.

(6) A poultice made in the ordinary way without precautions, covered with non-sterilized gauze. A culture from inner surface showed many confluent colonies of whitish color, which microscopically were seen to consist of short, thick bacilli, probably non-pathogenic.

(7) Poultice made as in No. 6, but heated for five minutes at a temperature somewhat above 212° F. A culture showed a few small, whitish colonies seen to consist of micrococci.

(8) December 12th. Men's surgical ward. Poultice made without precautions; cooked thirty seconds. A culture showed colonies of colon bacilli.

(9) Poultice cooked five minutes; otherwise same as No. 8. A culture showed a few colonies of colon bacilli.

(10) December 13th. Surgical out-patient dressing-room. Poultice made with aseptic precautions; sterile gauze, aseptic hands, etc.; cooked thirty seconds. A culture showed a pure growth of fine bacilli, but no pus cocci.

(11) The same as No. 10. Cooked five minutes. A culture showed various bacilli of different forms, but no pus cocci.

(12) Surgical private ward. The poultice was made with all aseptic precautions, and sterilized gauze was used. The meal was boiled for three minutes. A culture showed various organisms present.

(13) The same precautions as in No. 12. The poultice was boiled for ten minutes. A culture showed various organisms.

(14) Woman's surgical ward. The meal was boiled for three minutes and poured out. No gauze was used. A culture was sterile.

(15) Out-patient surgical dressing-room. The meal was boiled for three minutes. No gauze was used. A culture showed only one colony of long, thin bacilli.

(16) Surgical out-patient dressing-room. The poultice was made with all asceptic precautions, and the meal was boiled for two minutes. A culture showed long, thin bacilli and cocci.

(17) The poultice was made with all aseptic precautions, and was boiled for five minutes. A culture showed long, thin bacilli and cocci.

These experiments show plainly, that although apparently by chance the flaxseed-meal poultice may be perfectly sterile, yet in the majority of cases it is not so. Nor can it be rendered sterile by the most scrupulous care in its preparation — as shown, for example, in experiments Nos. 16 and 17. Experiments made in heating the meal for ten minutes or more reduced it to such a gelatinous mass that it was not suitable for surgical use, so that the times given here are as long a period of sterilization as is practicable. The organisms found were in most cases probably non-pathogenic and micrococci were not often present. The place

where the poultice was prepared seemed to have little effect on its character, experiments Nos. 12 and 13 having been made under the most favorable conditions, while experiment No. 1 was done in a ward containing many of the foulest cases of diphtheria.

In conclusion, it may be said that the flaxseed-meal poultice is a dressing often sterile, and, if not sterile, containing, as a rule, organisms which are probably not pathogenic; but that it may contain pus cocci. It may be added that no care in its preparation can be relied on to render it sterile.

The writer is indebted to Dr. Stokes, of the Pathological Department of the hospital, for his kind assistance.

XVI.

A CASE OF GENITAL MALFORMATION.

By John C. Munro, M.D.

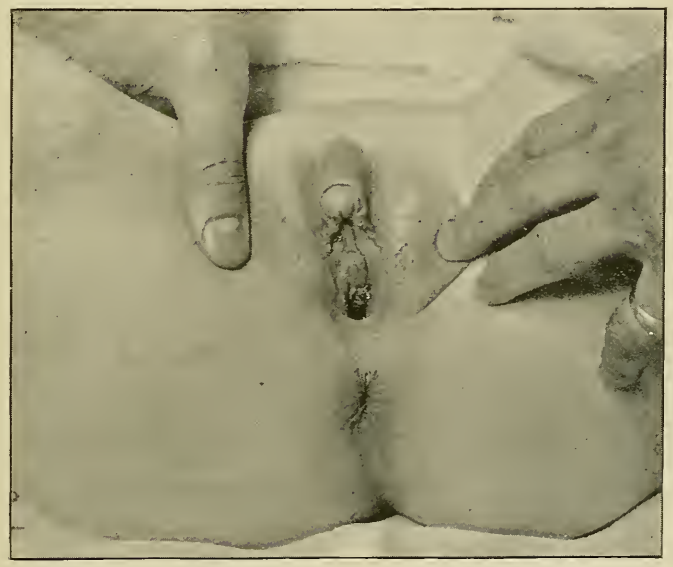
Isabelle ——, an English girl, was first examined in August, 1893, at the age of thirteen years and eleven months. She worked in a rope factory, was modest and domestic in her tastes, and came to the hospital on account of the non-appearance of the menses. Her feet and hands were rather large for a girl of her age, and there was a tendency to knock-knee.

The nipples were small and undeveloped; outside the areola, which was pinkish and half an inch in width, no glandular structure could be felt.

The inguinal canals were empty. The mons was well marked, and, like the labia majora, covered sparsely with black hairs, which on the labia were found to the edge of the mucous membrane, but from the mons did not extend upwards to the umbilicus.

The labia were two and one-half inches in length and three-fourths of an inch wide, but nothing suggesting a testis could be felt in them. The posterior commissure was well marked, and a very faint raphe could be seen on the perineum extending to the pigmented anus.

The clitoris, measuring on its dorsum one and a quarter inches, was apparently erectile and had a normal, slightly pigmented prepuce which below (posteriorly) merged into two mucous folds, the labia minora. The glans clitoridis



was well marked with a corona and cervix for four-fifths of its circumference, the lower fifth being flattened and slightly dimpled in the position of the male meatus. On the dorsum of the cervix was a delicate raphe. The glans as a whole, however, was smaller relatively to the rest of the organ than in a normal penis, and was noticeably pale. In the cervix smegma was found.

One-half inch below the clitoris was the meatus urinarius, the intervening space being marked in its lower portion by a depressed raphe. From the meatus two indistinct mucous folds passed downwards and outwards (posteriorly and laterally) for a short distance, to be lost in the lateral walls

of the vagina. Through the vagina the outer end of the urethra felt bulbous; its length was normal.

The hymen admitted a small finger. The vagina of normal depth contained no rugæ except on the left upper (anterior), and to a less extent on the right upper (anterior) wall. At the depth of the vagina was a crescentic fold lying in the coronal plane, the horns extending towards the vaginal opening for an inch or more; this mucous fold or shelf projected about a quarter of an inch, its median portion being occupied by a depression, puckered laterally, corresponding to the os uteri.

On bimanual examination, under ether, could be felt, both externally and internally, a body measuring from three-quarters to an inch transversely and about one-half an inch longitudinally, projecting as above intimated for about a quarter of an inch into the vagina. A sound could not be passed into this body.

To the right and in the normal position of an ovary could be felt a body of the size of a pea; on the left nothing could be felt.

The recto-vaginal area was normal. There were no indications of a prostate.

A year and a half after examination the patient had not menstruated; that is, when fifteen and a half years old.

XVII.

THE ROUTINE METHODS EMPLOYED FOR SECURING ASEPSIS IN THE SURGICAL WORK CARRIED ON IN THE AMPHITHEATRE, SMALL OPERATING ROOMS, AND WARDS OF THE SURGICAL DEPARTMENT OF THE BOSTON CITY HOSPITAL.

By Paul Thorndike, M.D.

The amount of surgery which is constantly being done in a great metropolitan hospital is so large, and is carried out by so many different surgeons and their assistants, that uniformity in the methods of work is an absolute essential. The writer believes that a short *résumé* of the details of the system in use in all aseptic work done in the surgical department of the hospital will be interesting enough to warrant its publication in the hospital's annual report. The following pages, therefore, are printed not with any claim for the perfection or originality of the methods in use, but merely as a description of them.

The surgery of the hospital is chiefly done in the large amphitheatre, one or two small operating rooms, and in the accident room. These rooms are close together, and near them all is the "sterilizing room," which is in the care of the operating-room nurse, and contains everything necessary for the sterilization of all instruments, garments, sheets, etc., which are to be used in operative work. This nurse is a graduate nurse who does no ward work, and who is in *permanent* charge of the operating rooms of the hospital. She has complete control of the sterilizing room, and is directly responsible for all work done in it. She has working under her direction a nurse pupil, who serves as her assistant for the term of one week, and in that time gets a good idea of the routine methods of work in the operating rooms

of the hospital. The nurse pupil is examined as to her proficiency in such work by the operating-room nurse at the close of her week's training.

THE OPERATING-ROOM PUPIL.

COURSE OF INSTRUCTION AND GENERAL REGULATIONS.

The nurse is to be notified, when possible, two weeks in advance, so as to have time for the course of reading and study as follows:

Prudden's "Story of the Bacteria."

Miss Hampton's "Notes on Nursing," Chapters 8, 9, 15, 17.

Billroth's "Care of the Sick," Chapter 4.

She is to be instructed by the operating-room head nurse in sterilization as practised in the sterilizing and operating rooms; the preparation of disinfectant solutions, dressings, instruments, sutures and ligatures, and everything required in operations, including the patient and nurse. She is to witness etherization and wait on the surgeons when practicable. At the end of the week following her course, she is to be examined by the operating-room head nurse, and a record and ranking of her efficiency and knowledge on the subject be reported in writing to the Superintendent of Nurses.

The course is arranged for one week. The hours on duty are from 7.30 A.M. to 8 P.M., with the time off duty allowed nurses in the wards. If any grave emergency occurs in the night, she is to be called to assist the Night Superintendent of Nurses, and allowed to sleep a sufficient time the next day to make up her rest. The hours for meals are:

Breakfast	7 A.M.
Dinner	1 P.M.
Supper	6 P.M.

Her uniform is to be scrupulously neat, and conform in every particular to the standard.

INSTRUMENTS.

Whenever possible, the instruments to be used are selected from the cases on the night before the time set for an operation, and are turned over to the operating-room nurse for sterilization. When this is not possible, they are sterilized while the other preparations for the operation are in progress, and in either case are prepared as follows:

Rules for Sterilizing Instruments. — Wrap the instruments selected for operations in two or three thicknesses of cotton cloths of appropriate shapes and sizes, in such manner as to exclude them from the air, and to make of them securely fastened compact bundles. (Knives and delicate sharp-edged instruments should each have their blades enveloped in separate layers of sheet wadding, and should be made up into bundles afterward, separate from the other instruments.)

When prepared, the bundles are to be placed in the sterilizer and subjected to dry heat of 120° C. [248° F.] for twenty minutes. The bundles are then to be removed and taken to the operating room as they are needed, but are not to be opened until ordered by the surgeons or their assistants, and after being opened the instruments are not to be handled by any one whose hands are not aseptic. If for any reason the instruments which have been sterilized for an operation are not used at the time intended, they should be taken from their bundles and returned to the instrument cases, after being carefully dried if moist. Once a bundle has been opened, the instruments it contains are to be considered non-sterile.

Warning. — Instruments with hard rubber or wooden handles or fittings, rubber and webbing catheters, bougies, and rubber articles of whatever sort are *not* to be subjected to heat.

SPONGES.

The sponges in common use are little gauze bags filled with cotton waste and then sterilized (see below). Reef

sponges are seldom if ever used, but are kept ready, and are prepared according to the following plan:

Cotton-Waste Sponges. — Place in cotton bags provided for the purpose. Sterilize for sixty minutes with dry heat in the thermostat at 120° C., then with moist heat for ten minutes at 120° C. [248° F.]. At the end of this time remove the bags, label them "Sterile" and store in glass jars until required for use. When needed, the sponges are to be removed from the jars and bags by sterilized forceps or sterilized hands only, and are not to be allowed to come in contact with any non-sterile objects. All cotton-waste sponges used in operations are to be destroyed thereafter; those not used are to be again sterilized before being used for another operation.

Reef Sponges are to be sterilized in four five-gallon covered earthen crocks, each of which should be provided with a wire basket which fits its interior and can be easily lifted out. Each basket should be provided with a perforated galvanized-iron diaphragm in the middle of which a short handle is inserted: the diaphragm should fit loosely the interior of the basket (the diaphragm lies on top of the sponges in the basket, and is to be used to press the fluid out of them when the basket is lifted from the crock). The crocks are to be marked A, B, C, and D respectively. Two, A and C, should contain solutions of carbolic acid, 1–20; two, B and D, solutions of carbolic acid, 1–80. Labels should also be placed on each jar to indicate the strength of the solution it contains.

Sponges which have been used in operations are not to be returned to the crocks until they have been thoroughly cleansed.

The crocks A and B are to be emptied of their solutions fortnightly, washed thoroughly with boiling water, and filled with fresh solutions of carbolic acid as before.

Reef sponges are mechanically cleaned and bleached in the Dispensary before coming to the sterilizing room, and any others offered are to be rejected.

To sterilize the sponges, place a basketful of them in crock A (1–20 carbolic-acid solution), allow them to remain in there for six days; then lift them out in the basket, squeeze

the fluid out of the sponges into Crock A, and place them, still in their basket, in Crock B (1–80 carbolic acid). Crocks C and D are provided with hoods. Crocks C and D are to be placed in shallow pans containing about two inches of water. The sponges in their basket as they are required for use are to be transferred from B to C in the same manner as described above; the hood is then placed over the outside of Crock C, its lower edge being beneath the water. The sponges are to be left in Crock C for six days, then transferred in the same way as before to Crock D. The sponges in Crock D, and Crock D only, are now ready for immediate use at any time. When required for operation, the basket is to be lifted from Crock D, the sponges squeezed by pressure with the "presser," then with sterilized forceps (*not* with the hands); as many as are needed are taken from the basket, placed in a sterilized cloth or bag, in which they should be securely enveloped and not uncovered until called for at operations.

CATGUT.

The gut used for sutures and ligatures is prepared in the chemical laboratory and furnished to the surgical department as needed. Mr. Greenleaf R. Tucker, the chemist of the hospital, has very kindly given me this description of the method of preparation, as devised and used by him.

Sterilization of Catgut. — "Catgut is sterilized in aniline oil. The gut is first soaked in the oil for from 12 to 24 hours, and the sterilization is completed by heating the gut and oil together at a constant temperature of 120° C. for fifteen minutes. The spools containing the gut are then transferred to alcohol and allowed to remain till the aniline oil is extracted. Finally, they are kept in alcohol with or without glycerine.

"This method when properly carried out is entirely successful, rendering all sizes of catgut sterile. Its efficiency is due to the fact that aniline oil is itself a germicide, that it is penetrating (*i.e.*, it permeates the gut through and through, without softening or otherwise altering its physical

condition), and that the gut so treated may be heated, without injury, to a temperature which is in itself sufficient to kill micro-organisms.

"At this hospital the method pursued is as follows: The catgut without preliminary treatment is wound upon glass spools and kept always in stock. Some spools are kept immersed in aniline oil in tightly covered glass specimen jars. When sterilized gut is wanted, some of these spools are removed from the jars, placed in a beaker, covered with aniline oil, and heated to 120° C. in a bath of aniline oil contained in a non-breakable vessel. A thermometer is kept immersed in the inner vessel (the beaker), and the contents stirred almost constantly. When the temperature reaches 115–120° C. the heat is carefully regulated so that the temperature may not rise above 120° C. After fifteen minutes' exposure to this temperature the spools are removed with sterile forceps and dropped into covered glass jars containing alcohol. This dissolves out the excess of aniline oil in a few hours, and the spools are then transferred to the glass receptacles in which they are kept in alcohol. If the gut has been slightly stiffened by the heat it is kept in alcohol containing five per cent. of glycerine, which brings it back to its normal condition."

NEEDLES.

Needles of such sizes and shapes as are required for an operation are threaded and then attached to sterilized towels. These are then folded, and each towel is completely enveloped in another sterilized towel, and securely fastened into a convenient bundle. These bundles are sterilized in the sterilizer for three-quarters of an hour in moist heat at 120° C. [248° F.], and are not opened until called for by the surgeon or his assistants. Once a bundle has been opened, the contents are considered non-sterile after the operation is completed, and the needles and sutures which have not been used, after being carefully dried are again sterilized before being used in another operation.

Care is taken that only sharp needles and strong sutures are selected for use.

DRAINAGE TUBES.

Drainage tubes of rubber are sometimes used and are kept constantly ready. They are prepared as follows:

Rubber tube for drainage, when new, is cut into suitable lengths, and cleaned as follows: each piece is slipped over a solid glass rod and scoured by means of a brush with soap and water. Then after rinsing in hot water, they are put into a wire cage and sterilized for one hour by moist heat at 120° C. The cage is removed from the sterilizer, and each piece of tubing lifted out by sterilized forceps and dropped into a glass specimen jar containing a solution of carbolic acid, 1–20, where they are kept for use. This jar is kept covered, except while the tubes are being put in or taken out.

LINEN.

The sheets, towels, and gowns, etc., which are for use in operations are all sterilized by moist heat at 120° C. [248° F.], for an hour and a half prior to an operation, in the sterilizer. These articles are made into convenient bundles before being placed in the sterilizer, by being rolled securely in sheets. The bundles are not opened except by order of the surgeons or their assistants. Bundles once opened are not used for other operations until again sterilized. Such of these sterilized bundles as are not used at once are stored unopened in the cases or drawers provided for them, but if not used for forty-eight hours, are again sterilized before being used for operations.

OPERATOR AND PATIENT.

The operator and his assistants wear operating gowns sterilized as above. The gowns are so made as to completely envelope the wearer from neck to feet. They button behind, fitting closely at the neck, and having strings to fasten the short sleeves tightly above the wearer's elbows. Of course they are changed after each operation.

The hands and arms of the operator and his assistants are scrubbed with soap, hot water, and a nail brush; then put through a saturated solution of permanganate of potash; then through a saturated solution of oxalic acid; and finally are

soaked in corrosive-sublimate solution, a large, porcelain pail of which is always at the operator's elbow during an operation.

Patients are prepared for operation in the wards before they are carried to the operating rooms. The area to be operated upon is shaved, if necessary, scrubbed with soap and hot water, and a wet corrosive dressing is applied over night. This is left in position until the patient is etherized and placed upon the operating table in the amphitheatre. The dressing is then removed, the parts scrubbed again, washed off with ether and then with corrosive-sublimate solution. After the seat of incision is surrounded by sterile sheets and towels, it is ready for operation.

SURGICAL DRESSINGS.

The materials for the dressings in common use in the hospital are gauze and sheet wadding, both of which are sterilized by dry heat in small tin boxes with tight-fitting covers, each box containing enough gauze or wadding for one small dressing. This work is done daily, and the tins distributed to the various parts of the surgical department.

In preparing the gauze, it is first dipped in a five per cent. solution of glycerine in water, then put through a clothes-wringer and dried. Then it is cut up into squares of the desired size, fitted into the tins, and sterilized by dry heat to 150° C. The glycerine dipping keeps the gauze soft and pliable, and prevents its stiffening when heated.

Each ward is provided with a truck or carriage which can readily be pushed about the ward on its rubber-tired wheels, and which contains on its shelves everything necessary for a complete aseptic dressing, — nail brushes, antiseptic solutions, sterile drainage-tubes, sterile dressings and bandages in abundance, and sterilized instruments. These instruments (scissors, forceps, dressing forceps, probes, directors, etc.) are sterilized each morning by the nurses in charge of the ward, by boiling. They are then wrapped in sterile cloths and put on the ward carriage ready for use.

The above details are all carefully compiled into a set of directions which are posted in the various operating rooms and the sterilizing room, for the use of the various people engaged in the work of the department. It seems hardly necessary to append these directions in full, as they have been essentially embodied in this paper.

XVIII.

STATISTICS OF 308 CASES OF SERIOUS INJURY TO THE HEAD.

Collected by E. H. Nichols, M.D.

The following 308 cases were treated as house patients at the Boston City Hospital, during the six years extending from Jan. 1, 1887, to Jan. 1, 1893. The cases fall naturally into the following classes:

	Cases.	Deaths.	Autopsies.
Concussion	78	5	2
Simple depressed fracture	10	5	2
Compound fracture without depression	42	8	3
Compound depressed fracture	74	22	6
Fracture of the base	82	60	14
Gun-shot injury	14	10	5
Miscellaneous	8	5	5
Totals	308	115	37

Taken collectively, of the 308 cases, 208 were between twenty and fifty-five years of age, 64 were between ten and twenty, and 32 were under ten years. The males out-numbered the females 272 to 36.

It is noteworthy that the character of the injury corresponded closely to the amount of violence exerted, as will be seen under the separate classifications. In two-thirds of the cases the external injury was in the parietal or frontal region.

The mental condition, too, was a good index of the severity of the injury. One hundred and fifty-three cases were brought in unconscious, and of these, 88, or a little more than one-half, died. Sixty-nine cases were delirious or semi-conscious, and of these 17 died. Eighty-six cases were clear mentally, and 10 died.

Of 54 cases in which there was no reaction of the pupils, 47 died.

Well-marked localizing paralysis was very rare, and very few operations were undertaken with paralysis as the only indication. Facial paralysis was the most common form of paralysis. Of 47 cases, 26 died.

Hæmorrhage from the ear was a common symptom in every class of injury. Of 74 cases, 43 died.

Incontinence or retention of urine as primary symptoms were not common, but the symptoms were of bad significance, as out of 45 cases, 27 died.

Subconjunctival ecchymosis occurred in but 16 cases, but of these 10 died.

Vomiting was an unfavorable symptom in all classes, except those classified as concussion. Of these latter, 23 had vomiting as a prominent symptom, and none died. Of those cases not classified as concussion, 35 had marked vomiting and 17 died.

Trephining was done in 24 cases, with 14 deaths; and in 20 cases fragments of bone were removed, with 4 deaths.

Excluding the cases of gun-shot injury which came to autopsy, and in all of which there was very extensive laceration and destruction of brain tissue with hæmorrhage, there were 32 autopsies. In 13 of those autopsies there was well-marked localized extradural hæmorrhage.

Consider each class of cases separately:

Concussion — 78 cases.

Under this heading are included those cases which had more or less prominent "head symptoms," — generally greater or less loss of consciousness with, in some cases, a certain degree of paralysis, and in which there was no evidence of fracture. Eleven cases only were under ten years of age. Twenty-three cases were under twenty, and 54 were between twenty and fifty-five, showing that the injury was not common in patients beyond middle age. The cause was chiefly slight falls, 51 of the cases occurring after falls less than twenty feet; most of them very much less. Two-thirds of the local injuries were in the parietal or

frontal region. Thirteen cases were conscious on arrival at the hospital, and 1 died. Thirty-six were delirious or semi-conscious, and 2 died; and 29 were unconscious, and 2 died. In 3 cases there was no reaction of the pupils, and 2 died. In 9 cases there was slight facial paralysis, and all recovered. In 1 case there was paralysis of the right arm and both legs, and death ensued in twelve hours. Subconjunctival ecchymosis was present in 1 case, and the patient recovered. Hæmorrhage from the ear occurred in 8 cases, with no deaths. Incontinence or retention occurred in 7 patients, and none died. The most notable symptom was vomiting, which was marked in 23 cases, and no one of the patients died. In 2 cases death was inside of twelve hours, while 3 lived from seven to nine days.

The 2 cases which came to autopsy showed well-marked œdema of the brain, without any evidence of fracture, contusion, or hæmorrhage, although 1 case was complicated with congestion of the lungs, following exposure. No bone operation was performed on any case.

SIMPLE DEPRESSED FRACTURE — 10 CASES.

One case was a child under ten years of age; 4 were between ten and twenty: and 4 between twenty and fifty-five. These cases all followed extreme violence, either falls from a great height, or blows on the head from some very heavy object. Six cases were brought in conscious, and 1 died; and 4 were unconscious, all of whom died. In 1 case the pupils did not react, and the patient died. In all the other cases the pupils were both equal and reacted. In 1 case there was right-sided hemiplegia, with injury in the left parietal region. One case had convulsions of the right arm and leg, following an injury just above the left ear, and died thirty-six hours after a trephining operation; and 1 patient with injury in the left temporal region had right-sided convulsions on the third day. Hæmorrhage from the ear was observed in but 1 case, and the patient died. Subconjunctival ecchymosis occurred in 2 cases, with 1 death. In 2 cases there was incontinence or retention, and both died. In 2 cases there was vomiting, with 1 death. One case with

hemiplegia of the side opposite the injury was trephined and at once showed immediate bulging of the brain and hernia cerebri, and died in an hour and a half. One patient was trephined on the second day for symptoms of increasing compression, the fragments were elevated, and the patient recovered. One patient was trephined to relieve epileptiform symptoms of several years' standing, following a blow on the head, and recovered from the operation, without relief of head symptoms. All but one of the fatal cases died within thirty-six hours.

There were 2 autopsies. In 1 case there was fracture of the right orbital plate, with local extradural hæmorrhage, and in the other there was contusion of the brain at the seat of the injury, with meningeal hæmorrhage.

COMPOUND FRACTURE WITHOUT DEPRESSION — 42 CASES.

These occurred mostly — 32 cases — in adults between twenty and fifty-five years of age. They were caused generally by comparatively slight violence; *e.g.*, 17 cases were cases of assault. The external injuries were chiefly in the parietal or frontal region. Twenty-two cases were conscious and none died. Eleven were delirious or semi-conscious and 4 died, while of 9 unconscious patients 4 died. Of 4 cases where the pupils did not react, 3 died. Three cases had definite localizing paralysis. In 1 case there was right-sided paresis, and the patient was trephined on the fifth day and an extradural clot removed. The patient died the afternoon of the same day. Another patient with left hemiplegia was trephined, the dura punctured, and a clot removed. The source of the hæmorrhage was not reached, and the patient died in eighteen hours. A third case with left hemiplegia was trephined over the middle meningeal artery, the clot removed and the meningeal artery tied, and the patient recovered in thirty-seven days. Hæmorrhage from the ear was seen in 7 cases and 4 died; and hæmorrhage from the nose in 3 cases and 2 died. Incontinence or retention of urine occurred in 5 cases and 4 died, while of 9 cases in which vomiting was a marked symptom 3 died. In 1 case fragments of bone were removed on the ninth day after the

injury, and the patient recovered. In 36 cases the wound was closed with antiseptic precautions, without any bone operation, and all but 4 healed by first intention.

There were 3 autopsies, in 2 of which there was fracture of the base, extending into the petrous portion of the temporal bone, with localized extradural clot at the base of the brain, while in the third case, after 6 weeks' illness, there was purulent meningitis and leptomeningitis, with multiple abscesses.

COMPOUND DEPRESSED FRACTURE — 74 CASES.

Forty-seven of these were between twenty and fifty-five years of age. Forty cases were caused by falls from distances exceeding twenty-five feet, or by railroad accidents. In this class of cases, also, eighty per cent. of the external wounds were in the frontal or parietal region. Thirty-four were conscious and 4 died; 9 were semi-conscious or delirious and 2 died; and 31 were unconscious and 16 died. In 16 cases there was no reaction of the pupils and 11 died. In 29 cases there was no paralysis, as expressly stated in the records; in 35 cases there was no mention made of any paralysis, and in 6 cases there was marked facial paralysis. In 3 cases, either on entrance, or in a comparatively few hours, there developed unilateral convulsions on the side opposite the injury, while in 1 case similar convulsions followed an injury over the vertex. Of these 4 latter cases 3 died. Hæmorrhage from the ear occurred in 5 cases with 3 deaths. In 9 cases there was hæmorrhage from the nose only, and 2 died; and in 1 case of hæmorrhage from the mouth only, the patient died. Subconjunctival ecchymosis occurred in 3 cases, with 2 deaths, and incontinence or retention of urine in 8 cases, with 3 deaths. In 15 cases fragments of bone were removed and 3 died, while 11 cases were trephined and 5 died. Of the latter, 1 operation was done on the fourth day for hemiplegia, and the patient recovered. One was done for increasing hemiplegia due, as shown by the autopsy, to purulent meningitis, and the patient died in eighteen hours. One was for the relief of marked hemi-

plegia. A large clot was removed and the middle meningeal artery tied, and the recovery was complete, although complicated by a large hernia cerebri; and in 1 case with profuse hæmorrhage, the posterior branch of the middle meningeal artery was tied, with recovery.

There were 6 autopsies. In 1 there was fracture of the base, with extradural clot at the base of the brain on the same side as the injury. In 3 cases there was fracture of the base with contusion of the brain. In 1 there was acute purulent meningitis, and in 1 acute softening. All but 1 of the cases died inside twenty-four hours. That one lived fifteen days, and died, as shown by the autopsy, of acute softening of the brain.

FRACTURE OF THE BASE — 82 CASES.

Fifty-six cases were between twenty and fifty-five years of age. The cause of the injury was variable, — in 56 cases from falls from heights varying from three feet to six stories. In many cases no external injury was obvious, but of 37 cases in which mention is made of external injury, 12 were in the occipital region. Sixty-two cases were unconscious, of whom 48 died. Fourteen were semi-conscious and 8 died; and 6 cases were conscious and 4 died. The pupils failed to respond in 24 cases, all of whom died. Hemiplegia was marked in 2 cases on entrance, and 1 recovered. Facial paralysis occurred in 26 cases, with no evidence of constant relation to the seat of the injury. Hæmorrhage from the ear occurred in 50 cases, with 33 deaths. Subconjunctival ecchymosis occurred in 6 cases and 4 died. Incontinence or retention of urine occurred in 14 cases with 12 deaths. Vomiting was marked in 10 cases and 6 died. One case was trephined over the right motor centre for symptoms of hemiplegia, and a large extradural clot was removed, but the artery was not tied. The patient died in twenty-one hours. One was trephined for symptoms of cerebral irritation and convulsions, but the source of the hæmorrhage was not found and the patient died. One patient was trephined over the mastoid region for symptoms of mastoiditis eight days after the injury, but

no abscess was found and the patient died seventeen days later.

There were 14 autopsies, in 5 of which there was well-marked extradural clot, with extensive fracture of the base. In 1 there was hæmorrhage into the ventricle from the rupture of an old apoplectic cyst; in 1 there were multiple capillary hæmorrhages, and in 1 acute softening. In addition to the head injury there was in 3 cases fracture of the cervical spine; in 2, compound fracture of the femur, and in 1, compound fracture of the leg, and in 1 other each, compound fracture of the elbow and of the humerus.

GUN-SHOT INJURY — 14 CASES.

One patient only was under twenty, and all were men. Two cases were accidental, the others suicidal. In all 14 cases the wound of entrance was on the right side. Twelve cases were completely unconscious. The 2 cases that came in conscious both recovered, and in neither case was there puncture of the dura. In 7 cases the pupils were equal and reacted, notwithstanding extensive laceration of the brain. In no case were the local symptoms so well marked as to be of any value in determining the course of the ball. There was subconjunctival ecchymosis in 2 cases and 1 died. Incontinence or retention of urine was well marked in 5 cases and 4 died. Vomiting occurred in 4 cases and 2 died. In 1 case the skull was trephined at a point directly opposite the point of entrance and the bullet removed from that point. The patient died in twelve hours, of shock. In 2 cases the wound was enlarged and bone removed, but the bullet was not found. One died and 1 recovered after four weeks. In 2 cases the wound was enlarged and the bullet removed from outside the dura, and both patients recovered. In 1 case there was a button removed at the point of entrance of the bullet and drainage established, the patient dying after three weeks, from exhaustion. In all cases which came to autopsy there was very extensive laceration and destruction of brain tissue, and hæmorrhage.

MISCELLANEOUS — 8 CASES

were so classified. Six were unconscious and 4 died; 1 was delirious and 1 died. The pupils failed to react in 3 cases and all died. One patient was trephined over the motor centre for the relief of an opposite hemiplegia, the brain was found disorganized, and the patient died twenty days later. One was operated on for mania of four years' standing, following a blow on the head. The patient recovered from the operation, but obtained no relief from the mental symptoms.

There were 5 autopsies: in 1 was found cerebral atheroma with diffused meningeal hæmorrhage; in 2, fracture of the base with contusion of the brain; in 2, fracture of the base with localized extradural hæmorrhage.

IN CONCLUSION.

The most unfavorable individual symptom was failure of the pupils to react. Subconjunctival ecchymosis was almost as unfavorable. Cases with retention or incontinence of urine occurring as a primary symptom, or with continued vomiting, terminated fatally in about one-half of the cases.

In but 12 cases were pressure symptoms so definitely localized as to be sufficient indication for operation. In the majority of cases, bone operations were done for the relief of the local symptoms; *i.e.*, to elevate depressed bone.

The cases show a larger proportion of localized extradural hæmorrhages than is generally appreciated, although often the location of the clot could not be exactly determined from the clinical symptoms.

Of 10 cases in which blood clot was removed, either after trephining or removal of bone fragments, without ligaturing the source of the hæmorrhage, but 2 recovered. The 3 cases in which large blood clot was removed and the bleeding vessel ligatured all recovered.

This suggests that it may be desirable in obscure head

cases to make an early rapid exploratory opening or openings in order to locate the probable hæmorrhage; and that it is desirable, in all cases where the clot is located, to enlarge the opening sufficiently to detect the source of the hæmorrhage, and to tie the bleeding vessel when found.

XIX.

TRAUMATIC GLYCOSURIA.

OBSERVATIONS MADE IN TWO HUNDRED AND TWELVE CASES OF HEAD INJURIES.

F. A. Higgins, M.D., and J. B. Ogden, M.D.

Ever since the time of Claude Bernard, who first experimentally demonstrated the existence of the so-called sugar centre in the floor of the fourth ventricle, head injuries have been recognized as playing a certain part in the etiology of some cases of diabetes. In addition to permanent glycosuria following traumatism of the head, there is another form observed by some writers and of which a number of cases have been reported. This last is merely transitory, and comes on directly after the traumatism or in a few days. On the other hand, the permanent form of glycosuria occurs at a period of weeks or even months afterwards.

In this article very little work has been directed towards this last form. The work recorded here was undertaken with the purpose of establishing whether any relation exists between various head lesions resulting from traumatism, and transitory glycosuria. It is obvious that this could be done only by following out a considerable number of cases such as would be seen in a large municipal hospital. Accordingly the work has been performed with the permission of the surgeons of the Boston City Hospital, upon all cases of head injuries, regardless of severity admitted to the hospital wards during the thirteen months beginning Dec. 1, 1893, and ending Jan. 1, 1895, comprising in all two hundred and twelve separate cases. The observations are the results of frequent and systematic examinations of the urine, obtained as soon as possible after the injury, and con-

tinuing from time to time during the patient's stay in the hospital. This was done in order to ascertain if any connection existed between the nature of the injury, whether mild or severe, and the appearance of sugar in the urine. Further, to see if such a condition, when it occurred, could render any assistance in diagnosis, especially in those not infrequent cases in which the diagnosis may be obscured or doubtful, such as many so-called fractures of the base of the skull, resulting in recovery with no other prominent symptoms than temporary unconsciousness and hæmorrhage from the ear. The pathology of the disease is not entered upon at all, but a number of cases in which autopsies were performed are reported in full.

First will be considered the literature of *permanent traumatic glycosuria*, and second that of *transitory glycosuria*.

Literature.

I. The literature of *permanent traumatic glycosuria* is comparatively meagre, yet traumatism is generally mentioned as one of the etiological factors in most works on diabetes.

A few examples of traumatic glycosuria are found in the works of Seegen, Fritz, Recklinghausen, Fischer, Mahe, Tod, Cyr, Itzigsohn, and Leyden. But Charcot is of the opinion that many of these are more often cases of persistent glycosuria than of true diabetes.

Lyman (1) says, "Injuries (and tumors), especially if they involve the fourth ventricle, are often accompanied by the development of diabetes." Strümpell (2), "Glycosuria may result from severe concussion of the brain and fracture of the skull." Tyson (3) says, "Excepting the injuries to the nervous system, which are known to cause diabetes, our knowledge of its causes is not precise. With regard to the the former, it is well known that blows upon the skull, with or without fracture, and concussions, communicated to the brain, spinal cord, and vaso-motor centres in other ways, as by falls, are such causes." Griesinger (4) found that thirteen out of two hundred and twenty-five cases which he collected were of traumatic origin. Luce (5) reports the case of a healthy lad who fell while skating, striking the back of his head heavily on the ice, dying shortly afterwards of diabetes. Friedrichs (6) out of a large

number could only trace eight cases of diabetes to traumatism. Baccelli (7) reports the case of a young man who became diabetic after a concussion of the brain. At the autopsy a necrobiotic point was found in the centre of the pons-Varolii. Vogel (8) reports a case of a fireman upon whose neck a beam fell. In a few weeks there followed a severe glycosuria ending in death. At the autopsy nothing abnormal was found in the brain, and it is thought that diabetes resulted from concussion of the spine. Senator (9) says that mechanical injuries are among the most important of exciting causes, such as concussion of the brain and spinal cord or even of the whole body. Glycosuria usually makes its appearance in a few hours or days after the infliction of the injury, rarely not until later. Lasegue (10) reports a case of glycosuria occurring three months after the accident, in a man who fell down-stairs, causing cerebral concussion. Bernstein-Kohan (11) out of forty-five cases traced twenty-five to head injuries, and reports that sugar does not always appear immediately after the injury. If the sugar appears early the condition is very apt to be mild. If late, it may be severe, and rapidly fatal. Spencer (12), "Injuries of the head and spinal cord are often the cause of sugar in the urine. The sugar may last for a few hours only, or for a few days. As a rule, it quickly disappears, but in some cases the sugar persists and the patients begin to be diabetic from the time of the injury." Brouardel and Richardiere (13), in an article on traumatic diabetes from a medico-legal point of view, state that traumatism is a well-recognized cause of diabetes, and that in a considerable number of cases in head injuries, examination of the urine reveals sugar when it had been before unsuspected.

This completes, in brief, a summary of the literature touching upon the subject of *permanent traumatic diabetes*, and is sufficient to establish the fact that traumatism does play a more or less important part in the etiology of diabetes. With respect to *traumatic glycosuria* of the *transitory form*, it has been the object of much less notice on the part of medical writers and investigators. In connection with this form, the work reported in this article has been principally performed. In the works of Alladoff,

Schiff, Pavey, and Eckhardt this subject receives but a passing notice.

Redard (14) says, "A glycosuria uniformly presents itself after all forms of traumatism of the head, whether accompanied by fracture or not. It may show itself almost immediately after an accident." He quotes eight cases where it appeared in two days, and a number of other cases where it appeared after a number of weeks. He affirms that sugar, if carefully sought for, is nearly always found.

Robin (15) reports a case of temporary glycosuria following cerebral concussion, sugar lasting four days.

De Pietro (16) says that after wounds and injuries of the head, glycosuria may last from two to eleven days, according to the severity of the lesion. That it appears later and lasts longer in fractures than in simple wounds.

Nuzzi (17) says that glycosuria is more or less constant after head injuries. The time of appearance and the quantity of glucose varies greatly. His results are generally negative. In one hundred and fifteen observations, on thirty-nine patients, he found glycosuria only three times.

Oppenheim and Eisenlohr (18) have observed the transitory form of glycosuria in a few cases.

Buch (19) reports twenty-eight cases with literature, in which injuries occurred to various parts of the head, the glycosuria following, being of the transitory form.

Lancereaux (20) says that glycosuria following traumatism is much less serious than the other varieties. It is temporary, it may disappear in a few days, and it never continues longer than a few months.

This concludes a short review of the literature of transitory glycosuria. It consists merely of mere mention of the existence of such a condition by some writers, and reports of a limited number of cases by others. There seems to have been no attempt made to investigate any considerable number of cases with head injuries, or to trace any connection between the glycosuria and the nature of the head injury. Redard attempted to find a connection between the appearance of pus in the wound of the head and glycosuria, but that any connection between the two existed was denied by De Pietro.

CLASS I. — SIMPLE SCALP WOUNDS AND MINOR HEAD INJURIES. — *Concluded.*

No.	Name. Age. Sex.	Service.	Day of Injury before Entrance.	Cause of Injury.	Visible Lesion.	Symptoms.	Urine.			Day of Discharge. Result.
							Sugar.	Day of first Appearance after Injury.	Day of Disappearance after Injury.	
76	J. H. G. 36 yrs. M.	Monks.	Same day.	Thrown from wagon.	Scalp wound, rt. frontal region, 2 inches.	None.	None.	. . .	. . .	7th day. Rel'd.
77	J. McG. 35 yrs. M.	Monks.	"	Hit with stone.	Scalp wounds, rt. frontal and lt. parietal region.	"	"	. . .	. . .	10th " "
78	L. M. 50 yrs. M.	Cushing.	"	Struck by a box.	Scalp wound, 3 inches, rt. parietal region.	"	"	. . .	. . .	8th " "
79	E. C. 31 yrs. M.	Cushing.	Four days.	Fell on ice.	Scalp wound, $\frac{3}{4}$ inch, lft. supra orbital ridge.	"	"	. . .	. . .	10th " "
80	D. S. 30 yrs. M.	Cushing.	Same day.	Assaulted on head.	Scalp wound, occiput, compound fracture nose.	"	"	. . .	. . .	Still in hospital.
81	F. M. 25 yrs. M.	Monks.	"	Kicked on head.	Scalp wound, rt. parietal region, 2 inches, compound fracture inf. maxillary.	"	"	. . .	. . .	2d " "
82	R. C. 30 yrs. M.	Cushing.	"	Fell from horse.	Scalp wound, lft. frontal region.	"	"	. . .	. . .	3d " "
83	D. S. 20 yrs. F.	Cushing.	"	Struck by an officers' club.	2 scalp wounds, 2 inches, lft. parietal region.	"	"	. . .	. . .	4th " "
84	H. D. 54 yrs. M.	Cushing.	One day.	Fell on sidewalk.	Scalp wound, 3 inches, vertex.	"	"	. . .	. . .	4th " "

No.	Name. Age. Sex.	Service.	Day of Injury before Entrance.	Cause of Injury.	Visible Lesion.	Symptoms.	Urine.			Day of Discharge. Result.
							Sugar.	Day of first Appearance after Injury.	Day of Disappearance after Injury.	
51	M. D. 43 yrs. M.	Watson.	Same day.	Thrown from wagon.	Several scalp wounds, 1 to 3 in. long, on rt. temporal and parietal regions.	None.	None.	. . .	. . .	21st day. Rel'd.
52	J. O'B. 47 yrs. M.	Watson.	"	Fell 10 feet from roof.	Scalp wound, lft. parietal eminence, 1½ inch.	"	Trace.	18 hours.	Only time found.	7th " "
53	T. V. 23 yrs. M.	Watson.	"	Fell from top of freight car.	Scalp wound over bregma.	"	None.	. . .	. . .	2d " "
54	M. H. C. 37 yrs. M.	Watson.	"	Fell on sidewalk.	Scalp wound, occiput, 4 inches long.	"	"	. . .	. .	3d " "
55	N. L. 41 yrs. F.	Cheever.	"	Fell 20 feet on to sidewalk.	Scalp wound rt. frontal eminence, 1½ inch, and fractured hip.	"	"	. . .	. . .	Still in hospital.
56	C. C. 25 yrs. M.	Cheever.	"	Fell 20 feet into dry dock.	Scalp wound, occiput, 1½ inches, and fractured arm.	"	"	. . .	. . .	" "
57	J. C. G. 52 yrs. M.	Gavin.	"	Fell from electric car.	Contusion and hematoma, lft. frontal eminence.	"	None.	. . .	. . .	3d day. Rel'd.
58	R. W. 39 yrs. M.	Gavin.	"	Fell 15 feet into hatchway.	Several lacerated wounds about face and scalp.	"	"	. . .	. . .	Still in hospital.
59	B. C. 28 yrs. F.	Mooks.	"	Assaulted on head.	Scalp wounds, lft. frontal region, 2 inches.	"	"	. . .	. . .	6th day. Rel'd.
60	O. H. E. 46 yrs. M.	Mooks.	"	Assaulted on head.	Rt. parietal region, two wounds, 2 inches.	"	"	. . .	. . .	5th " "
61	J. G. 24 yrs. M.	Monks.	One day.	Fell into elevator well.	Scalp wound, rt. frontal eminence, 1½ inch.	"	"	. . .	. . .	14th " "
67	W. R. 36 yrs. M.	Monks.	"	Fell downstairs.	Scalp wound, occiput, 1 inch.	"	"	. . .	. . .	7th " "
63	J. M. C. 25 yrs. M.	Monks.	Two days.	Run over by a team.	Scalp wound, rt. parietal, 2 inches.	"	"	. . .	. . .	8th " "
64	H. McC. 39 yrs. M.	Monks.	Same day.	Knocked down by horse.	Scalp wounds, rt. parietal region.	"	"	. . .	. . .	6th " "
65	H. D. 40 yrs. M.	Cheever.	Two days.	Kicked on head.	Small wounds and contusions on head, compound fracture nose.	"	"	. . .	. . .	8th " "
66	J. E. W. 25 yrs. M.	Cheever.	Same day.	Fell from building.	Wounds of cheek and frontal region.	"	"	. . .	. . .	12th " "
67	D. M. 70 yrs. M.	Cheever.	"	Fell downstairs.	Scalp wound, 4 inches, occiput.	"	"	. . .	. . .	6th " "
68	J. E. W. 20 yrs. M.	Bolles.	"	Struck by file.	Scalp wound, 2½ inches, over sagittal suture.	"	"	. . .	. . .	4th " "
69	J. T. 31 yrs. M.	Gavin.	"	Thrown out of carriage.	Contusion, rt. parietal region.	"	"	. . .	. . .	6th " "
70	J. M. M. 25 yrs. M.	Gavin.	"	Assaulted on head.	Scalp wound, rt. frontal region, injury of eye.	"	"	. . .	. . .	14th " "
71	J. J. 6 yrs. M.	Gavin.	One week.	Fell on stove.	Scalp wound, 1 inch, occiput.	"	"	. . .	. . .	12th " "
72	A. C. 50 yrs. M.	Gavin.	Same day.	Kicked on head.	Scalp wound, ¾ inch, rt. frontal region.	"	"	. . .	. .	12th " "
73	P. F. 20 yrs. F.	Gavin.	"	Jumped 30 feet from 3d story window.	Scalp wound, 1 inch, lft. frontal region.	"	Present.	Trace, 24h after.	Six days after 18	6th " "
74	E. M. S. 38 yrs. F.	Burrell.	"	Assaulted on head.	Hematoma, lft. parietal and temporal region.	"	"	Trace, 10 hrs.	10th day	Still in hospital.
75	J. K. 51 yrs. M.	Monks.	"	Struck by flat-iron.	Scalp wounds, frontal region, 1 inch.	"	None.	. . .	.	14th " "

CLASS I.—SIMPLE SCALP WOUNDS AND MINOR HEAD INJURIES.—*Continued.*

No.	Name. Age. Sex.	Service.	Day of Injury before Entrance.	Cause of Injury.	Visible Lesion.	Symptoms.	Urine.			Day of Discharge. Result.
							Sugar.	Day of first Appearance after Injury.	Day of Disappearance after Injury.	
26	M. J. C. 33 yrs. M.	Burrell.	Same day.	Struck by derrick boom.	Scalp wound, rt. temporal region.	None.	None.	. . .	. . .	5th day. Rel'd.
27	W. E. T. 4½ yrs. M.	Post.	"	Run over by express wagon.	Hæmatoma, rt. parietal. Lacerated wound, ear.	"	Present.	9½ hrs.	Fifth day.	13th " "
28	M. E. 40 yrs. M.	Cushing.	Three days.	Assaulted with block of wood.	Scalp wound, 1½ inches long, vertex.	"	None.	. . .	. . .	16th " "
29	J. K. 29 yrs. M.	Burrell.	Same day.	Fell from herdle.	Scalp wound, rt. frontal region.	"	"	. . .	. . .	6th " "
30	A. M. 25 yrs. F.	Burrell.	"	Fell from electric car.	Scalp wound, rt. parietal region.	"	"	. . .	. . .	7th " "
31	M. E. 40 yrs. M.	Cushing.	Five days.	Struck on head by block of wood.	Scalp wound, rt. parietal region, 1½ inches.	"	"	. . .	. . .	16th " "
32	J. P. 26 yrs. M.	Post.	Same day.	Thrown from carriage.	Scalp wound, 1½ inches, vertex.	"	"	. . .	. . .	5th " "
33	H. I. 60 yrs. M.	Smith.	"	Fell on sidewalk.	Scalp wound, rt. frontal region.	"	"	. . .	. . .	5th " "
34	J. M. 29 yrs. M.	Smith.	One day.	Fell from wharf, 12 feet.	Scalp wound, lft. parietal region, 3 inches.	"	"	. . .	. . .	8th " "
35	A. D. S. M.	Smith.	Same day.	Unknown.	Scalp wound, frontal region, 2 inches.	"	"	. . .	. . .	2d " "
36	C. W. 33 yrs. M.	Smith.	"	Struck on head by a falling plank.	Scalp wound, 1½ inches, over vertex.	"	"	. . .	. . .	4th " "
37	J. G. 48 yrs. M.	Smith.	"	Fell on sidewalk.	Scalp wound, 2½ inches, over sagittal suture.	"	"	. . .	. . .	16th " "
38	W. D. 34 yrs. M.	Smith.	"	Fell off wagon.	Scalp wound, 1 inch, temporal region.	"	"	. . .	. . .	7th " "
39	P. M. D. 21 yrs. M.	Smith.	"	Fell from staging, 18 feet.	Scalp wound, 2 inches, rt. parietal region.	"	"	. . .	. . .	4th " "
40	P. C. 22 yrs. M.	Smith.	One day.	Stabbed.	Scalp wound, rt. parietal region.	"	"	. . .	. . .	5th " "
41	A. C. 34 yrs. M.	Smith.	Same day.	Assaulted on head.	Scalp wound, 3 inches, frontal bone.	"	"	. . .	. . .	3d " "
42	T. J. H. M.	Monks.	Same day.	Fell on curb-stone.	Scalp wound, occiput.	"	"	. . .	. . .	2d " "
43	F. H. 9 yrs. M.	Gavin.	"	Fell from wagon.	Scalp wound, lft. temporal bone.	"	"	. . .	. . .	14th " "
44	J. J. D. 33 yrs. M.	Gavin.	"	Assaulted on head.	Scalp wound, 1½ inch, rt. frontal bone.	"	"	. . .	. . .	8th " "
45	F. B. 23 yrs. M.	Gavin.	"	Fell from wagon.	Scalp wound, lft. frontal bone.	"	"	. . .	. . .	3d " "
46	J. K. 5 yrs. M.	Gavin.	"	Fell on curb-stone.	Scalp wound, med. frontal bone.	"	"	. . .	. . .	3d " "
47	J. M. 40 yrs. M.	Monks.	"	Thrown from wagon.	Scalp wound, rt. parietal, 7 inches.	"	"	. . .	. . .	7th " "
48	A. C. 40 yrs. M.	Monks.	"	Assaulted on head.	Scalp would, lft. frontal, 2 inches.	"	"	. . .	. . .	5th " "
49	H. G. 46 yrs. M.	Monks.	Two days.	Fell against brick wall.	Scalp wound, lft. frontal.	"	"	. . .	. . .	7th " "
50	C. H. 23 yrs. F.	Watson.	Same day.	Fell down stairs.	Scalp wound, rt. occiput, 1 inch.	"	"	. . .	. . .	6th " "

Class I. — Simple Scalp Wounds and Minor Head Injuries.

No.	Name. Age. Sex.	Service.	Day of Injury before Entrance.	Cause of Injury.	Visible Lesion.	Symptoms.	Urine.			Day of Discharge. Result.
							Sugar.	Day of first Albumen after Injury.	Day of Disappearance after Injury.	
1	W. E. 64 yrs. M.	Watson.	Same day.	Fell from electric car.	Scalp wound, rt. parietal region.	None.	None.	. . .	. . .	3d day. Rel'd.
2	A. L. 21 yrs. F.	Bolles.	"	Struck on forehead by cup.	Scalp wounds, frontal region.	"	"	. . .	. . .	4th " "
3	G. M. 31 yrs. M.	Watson.	Two days.	Fell on sidewalk.	Scalp wound.	"	"	. . .	. . .	4th " "
4	L. B. 40 yrs. F.	Bradford.	Three days.	Knocked down and struck on head by sled.	Scalp wound, rt. occipital region.	"	"	. . .	. . .	6th " "
5	S. D. 23 yrs. F.	Watson.	Two days.	Fell down stairs.	Scalp wound, lft. parietal region.	"	"	. . .	. . .	26th " Well.
6	P. M. 26 yrs. M.	Watson.	Fourteen days.	Fell down stairs.	Scalp wound, rt. frontal region.	"	"	. . .	. . .	5th " Rel'd.
7	C. J. S. 54 yrs. M.	Bradford.	Same day.	Struck on head with an unknown implement.	Scalp wound, lft. frontal region.	"	"	. . .	. . .	8th " "
8	L. P. 24 yrs. M.	Bradford.	"	Knocked down by a carriage.	Scalp wound, rt. temporal region.	"	"	. . .	. . .	2d " "
9	I. McM. 62 yrs. M.	Bradford.	"	Thrown from sleigh.	Scalp wound, median frontal region.	"	"	. . .	. . .	3d " "
10	J. M. 42 yrs. F.	Bolles.	"	Fell on pavement.	Scalp wound, 2 inches on vertex.	"	"	. . .	. . .	11th " "
11	F. M. 38 yrs. M.	Bolles.	"	Fell from moving train.	Scalp wound, rt. temporal region.	"	"	. . .	. . .	2d " "
12	D. C. 56 yrs. M.	Watson.	"	Fell, striking against corner of door.	Large hæmatoma over rt. temporal and frontal regions.	"	"	. . .	. . .	23d " "
13	J. H. F. 40 yrs. M.	Watson.	"	Fell on pavement.	Scalp wound, 1½ inches, lft. parietal region.	"	"	. . .	. . .	2d " "
14	B. S. 21 yrs. M.	Watson.	One day.	Kicked on head.	Scalp wound, rt. parietal region.	"	"	. . .	. . .	7th " "
15	M. K. 35 yrs. M.	Bradford.	Same day.	Assaulted on head.	Scalp wound, rt. frontal region.	"	"	. . .	. . .	3d " "
16	P. F. 20 yrs. M.	Watson.	"	Fell from wagon.	Scalp wound, rt. parietal region.	"	"	. . .	. . .	21st " "
17	B. H. 56 yrs. M.	Bradford.	"	Knocked down on pavement.	Scalp wound, rt. occipal region.	"	"	. . .	. . .	8th " "
18	J. K. 28 yrs. M.	Bolles.	Three days.	Knocked down.	Scalp wound, rt. temporal region, 1½ inches.	"	"	. . .	. . .	8th " "
19	J. G. 80 yrs. M.	Watson.	Same day.	Fell on sidewalk.	Scalp wound, rt. occipital region.	"	"	. . .	. . .	27th " "
20	C. G. 60 yrs. F.	Watson.	"	Fell down stairs.	Scalp wound, rt. parietal region, 1¼ inches.	"	"	. . .	. . .	3d " "
21	T. R. 26 yrs. M.	Post.	"	Assaulted on head.	Scalp wound, rt. parietal region, 2 inches.	"	"	. . .	. . .	7th " "
22	M. F. A. 57 yrs. M.	Burrell.	"	Fell down stairs.	Scalp wound, lft. parietal region, 7 inches.	"	"	. . .	. . .	9th " "
23	F. W. M. 30 yrs. M.	Post.	"	Fell on sidewalk.	Scalp wound, 2 inches, vertex.	"	"	. . .	. . .	3d " "
24	J. D. 40 yrs. M.	Cushing.	Nine days.	Fell against iron bar.	Scalp wound, 3 inches, rt. parietal region.	"	Present.	Twelfth day.	Fourt'nth day.	15th " "
25	D. M. 42 yrs. F.	Burrell.	Same day.	Assaulted on head.	Several scalp wounds, frontal eminence.	"	None.	. . .	. . .	20th " "

The tables which follow show the results of the observations on two hundred and twelve cases. They are arranged in classes according to the nature of the head injury, without regard to lesions in other parts of the body; yet whenever there have been such lesions, they have been noted both in the tables and in the description of individual cases.

Class I. includes the simple scalp wounds and other minor head injuries.

Class II., scalp wounds with bone exposed.

Class III., cases of concussion, meaning those followed by loss of consciousness without fracture.

Class IV. includes fractures of the vault of the skull.

Class V., fractures of the base of skull.

Class I.

Case 24, Class I. — J. D., male, aet. 40, laborer. Service of Dr. Cushing. Nine days before entrance was struck on the head by an iron bar, sustaining a scalp-wound three to four inches long. There were ten stitches taken in the wound at that time. Three days later erysipelas of the face and scalp developed. On entrance, beside the erysipelas, a discharging sinus was found at one point in the unhealed scalp-wound. The patient was discharged on the fifteenth day, the erysipelatous blush having entirely disappeared, and only a very slight discharge from the wound.

Urine. — The urine was examined the day following entrance, but no sugar found. Two days later (twelfth day after injury and ninth day after the appearance of erysipelas) the urine had a sp. gr. of 1020 and contained a slight trace of sugar, it being, however, too slight to quantitate. This trace of sugar continued for two days, then disappeared, and was not found again during his stay in the hospital.

There is obviously some doubt as to the cause of the sugar in this case. It appeared for the first time twelve days after the injury, and at a time when the erysipelas was rapidly improving. It would appear, however, as if the erysipelas was an exciting factor in the production of

the sugar, yet it may have been due directly to the injury, even though the sugar did not appear for twelve days. Brouardel and Richardiere report 4 cases of injury to the head where the sugar did not appear until from one to two weeks after the injury.

Case 27, Class I. — W. E. T., male, aet. $4\frac{1}{2}$. Service of Dr. Post. Was run over by an express wagon, and on entrance was found to have a hæmatoma over the left frontal eminence, besides a lacerated wound of left ear and contusion of left shoulder. The wound of the ear was dressed antiseptically. At no time were there any head or nervous symptoms. The patient was discharged on the twelfth day, apparently in excellent condition.

Urine. — The urine, obtained nine and one-half hours after the injury, was found to have a sp. gr. of 1035; albumen, the slightest possible trace; sugar, 2.93%. Fermentation by yeast was very active. Two days later the urine had a sp. gr. of 1035; albumen, slightest possible trace; sugar, a very faint trace. A specimen obtained on the fifth day after the injury showed a complete disappearance of the sugar. Repeated examinations of the urine, while the patient remained in the hospital, failed to show any trace of sugar after the fifth day.

Case 52, Class I. — J. O'B., male, aet. 47, janitor. Service of Dr. Watson. The patient while intoxicated fell from a roof, a distance of ten feet, sustaining a scalp wound one and one-half inches long, running transversely between the left parietal eminence and the median line. Several contusions about the left shoulder and right side. The pupils were equal and reacted to light. Mental state dubious, because of the alcoholic condition. Scalp wound sutured and dressed antiseptically. The following day the mental condition was good, and he was feeling very well except for lameness resulting from the fall. The patient was discharged well on the seventh day.

Urine.—The urine which was passed about eighteen hours after the injury had a trace of sugar, this being the only time that it was found. Three months after the injury a specimen of urine was obtained from the patient and found to contain no sugar.

Case 73, Class I. — P. F., aet. 20, female. Service of Dr. Gavin. Was confined in November, 1894. While suffering from puerperal mania, which developed fifteen days later, she jumped from a third-story window (30 feet) striking on the ground. She was brought to the hospital suffering from an incised wound of left super-orbital ridge, three-fourths of an inch in length, bone not exposed. Also on the inner aspect of the left breast a small wound one-half inch long and one-fourth inch deep. Milk expressed from both breasts. The wounds were dressed antiseptically. For the first few days the breasts were relieved of their secretion by means of a breast pump. The patient remained delirious during her stay in the hospital. The wounds entirely healed, so that she was discharged the sixth day.

Urine. — On the day after entrance a trace of sugar was found in the urine. The amount of sugar increased considerably in the next few days, for on the sixth day after entrance nearly 1% was found. There was not only a reduction of copper in Fehling's solution, but also a noticeable fermentation with yeast.

Case 74, Class I. — E. M. S., aet. 38, female. Service of Dr. Burrell. Was confined in March, 1894. Nine months later was struck on the head and face in a quarrel with her husband. On examination she was found to have a large hæmatoma over the left parietal and frontal eminences and extending down on to the left cheek. There was marked swelling of nose and about left eye, sufficient to close it. A small contused wound behind left ear, also on left cheek. The arms, legs, and lower part of trunk were covered with small excoriations and bruises. There was also a fracture of the second phalanx of the right ring finger. There was no paralysis or head symptoms. As the patient had been nursing her child up to the time of entrance to the hospital, the breasts required massage during the first twenty-four hours only, there being no trouble afterward.

Urine. — The first specimen of urine, obtained about twelve hours after entrance, was found to have a sp. gr. of 1018, large trace of albumen, and to contain a slight trace of sugar. After forty-eight hours the sugar had increased to a distinct trace, yet too slight to be quantitated, the urine

having a sp. gr. of 1026, and containing a very slight trace of albumen. There was a definite fermentative reaction obtained by yeast. An examination of the urine made ten days after entrance showed a sp. gr. of 1023, a very slight trace of albumen, and a complete disappearance of the sugar. Repeated examination, made later, failed to show the presence of sugar.

(Patient still in the hospital, at the time of writing.)

In the consideration of these last two cases we are confronted by the question : Have we to do with a lactosuria or a temporary glycosuria as a result of injury to the head and a general "shaking up"? In both cases, each was nursing a young child up to the time of injury, and according to Ord (21), lactose appears in the blood during lactation. Blot (22), De Sinetz (23), Hoffmeister (24), Kaltenbach (25), Halliburton (26), and Hammersten (27) say that lactose appears in the urine, as such, although Ord, Moritz (28), and Voit (29) seem to have found it appearing in the urine as glucose, yet Moritz (30) later asserts the probability of a lactosuria.

In distinguishing between glucose and lactose we find that, according to Hammersten, the latter is not fermentable with pure yeast, yet both reduce copper. If, then, this be true, although the non-fermentation of lactose is disputed by Halliburton, we may have to do, in these two cases, with a glycosuria, either alone or coincident with a lactosuria.

Class II.

Case 87, Class II. — D. C., male, aet. 29. Service of Dr. Bolles. Fell twenty-five feet from staging striking on right side of face, causing a wound three inches long, beneath which the malar bone, zygoma, and the coronoid process of jaw were fractured. In addition there was a small wound one inch long, with bone exposed in middle of forehead, and a Colles' fracture of left wrist. Patient was unconscious, remaining so several hours, following which was delirium, lasting most of the time till death; pupils reacting, but unequal; no paralysis. In dressing, a number of small pieces of bone from the face

Class II. — Scalp Wounds. Bone Exposed.

No.	Name. Age. Sex.	Service.	Day of Injury before Entrance.	Cause of Injury.	Visible Lesion.	Symptoms.	Urine.			Day of Discharge. Result.
							Sugar.	Day of first appearance after injury.	Day of disappearance after injury.	
85	E. F. 34 yrs. M.	Watson.	Same day.	Struck on head with hammer.	Scalp wound, frontal region. Bone exposed.	None.	None.	. . .	. . .	10th day. Rel'd.
86	W. B. 65 yrs. M.	Watson.	"	Struck on head with an axe.	Numerous scalp wounds rt. frontal and parietal region. Bone exposed and chipped.	"	"	. . .	. . .	45th " "
87	D. C. 29 yrs. M.	Bolles.	"	Fell from staging 25 feet, striking on face.	Wounds of rt. side of face and rt. temporal region. Fract. Zygoma and malar bone.	Unconscious for several hours, followed by delirium till death. Right facial paralysis.	Present.	12 hrs.	6th day.	8th " Died. Autopsy.
88	M. McD. 36 yrs. F.	Bradford.	"	Struck by an unknown implement.	Scalp wound, Med. frontal region. Bone exposed.	None.	None.	. . .	. . .	4th " Rel'd.
89	T. P. S. 34 yrs. M.	Bradford.	"	Struck on forehead by an unknown implement.	Scalp wound, frontal region. Bone exposed.	"	"	. . .	. . .	4th " "
90	M. F. 32 yrs. M.	Bolles.	"	Kicked on head.	Scalp wound, lft. frontal eminence. Bone exposed.	"	"	. . .	. . .	2d " "
91	D. O'B. 26 yrs. M.	Bolles.	Eight days.	Struck on head by brick.	Scalp wound, lft. parietal eminence. Bone exposed.	"	"	. . .	. . .	17th " "
92	P. H. 31 yrs. M.	Watson.	Same day.	Fell from wagon.	Scalp wound 9 inches long, rt. frontal-parietal region. Bone exposed.	"	"	. . .	. . .	14th " "
93	A. B. 43 yrs. M.	Bolles.	"	Fell from electric car.	Scalp wound, lft. parietal region, 7 inches long. Bone exposed.	"	"	. . .	. . .	6th " "
94	M. S. C. 63 yrs. F.	Bolles.	"	Fell from electric car.	Scalp wound, frontal region. Bone exposed and roughened.	"	"	. . .	. . .	4th " "
95	J. G. 4½ yrs. M.	Bradford.	"	Fell on sidewalk.	Scalp wound, 1½ inches, rt. parietal. Bone exposed.	"	"	. . .	. . .	2d " "
96	H. L. 17 yrs. M.	Post.	"	Struck by piece of falling iron.	Scalp wound. Bone exposed.	"	"	. . .	. . .	2d " "
97	T. H. 43 yrs. M.	Burrell.	"	Struck by piece of falling wood.	Scalp wound, vertex, 3 inches. Bone exposed.	Semi-conscious about half an hour.	"	. . .	. . .	7th " "
98	J. P. F. 41 yrs. M.	Burrell.	"	Bullet wound of head.	Perforating wound, occiput. Bone exposed. Bullet flattened against skull.	" " "	"	. . .	. . .	8th " "
99	T. F. 24 yrs. M.	Burrell.	"	Fell from top of freight car.	Scalp wound, occiput, 2 inches. Bone exposed.	Semi-conscious.	"	. . .	. . .	8th " "
100	J. S. 51 yrs. M.	Watson.	"	Fell 23 feet from staging.	Scalp wound, frontal region, 4 inches. Bone exposed.	"	"	. . .	. . .	8th " "
101	M. N. 32 yrs. F.	Watson.	"	Fell downstairs.	Scalp wound, rt. frontal region. Bone exposed.	"	"	. . .	. . .	8th " "
102	W. F. 11 yrs. M.	Post.	"	Struck on head by batted ball.	Rt. frontal region, scalp wound 1½ inches. Bone exposed.	"	"	. . .	. . .	5th " "
103	T. V. 21 yrs. M.	Monks.	"	Struck by tumbler.	Scalp wound 2 inches, rt. frontal region. Bone exposed.	"	"	. . .	. . .	3d " "
104	M. K. 8 yrs. M.	Smith.	"	Struck by brick.	Scalp wound 1 inch, lft. temp. region. Bone exposed.	"	"	. . .	. . .	2d " "
105	J. M. 40 yrs. M.	Post.	"	Thrown from wagon.	Scalp wound 2 inches, rt. parietal region. Bone exposed.	"	"	. . .	. . .	17th " "
106	A. M. E. 30 yrs. M.	Smith.	"	Struck by mallet.	Scalp wound, 1½ inches, over occiput. Bone exposed.	"	"	. . .	. . .	2d " "
107	G. J. T. 24 yrs. M.	Monks.	"	Assaulted on head.	Scalp wound, 2 inches, lft. parietal region. Bone exposed.	"	"	. . .	. . .	4th " "

No.	Name. Age. Sex.	Service.	Date of Injury before Entrance.	Cause of Injury.	Visible Lesion.	Symptoms.	Urine.			Day of Discharge. Result.
							Sugar.	Day of first appearance after injury.	Day of disappearance after injury.	
108	J. D. 50 yrs. M.	Gavin.	Same day.	Fell from electric car.	Scalp wound, rt. frontal region. Bone exposed and roughened.	Semi-conscious.	None.	. . .	. . .	3d day. Rel'd.
109	T. G. 74 yrs. M.	Gavin.	"	Fell from wagon.	Scalp wound, 4 inches, lft. frontal region. Bone exposed.	"	"	. . .	. . .	24th " "
110	H. D. R. 27 yrs. M.	Monks.	"	Struck on head by a descending elevator.	Scalp wound 3½ inches, occiput. Bone exposed, and compound fracture nose.	"	Neg.	. . .	. . .	8th " "
111	E. R. 56 yrs. M.	Monks.	"	Struck by a club.	Scalp wound 3 inches, lft. frontal region. Bone exposed.	"	"	. . .	. . .	2d " "
112	J. M. 50 yrs. M.	Monks.	Five days.	Fell from electric car.	Scalp wounds, frontal and lft. parietal regions, 2 and 2½ inches Bone exposed.	"	Present.	12 hours.	To death.	20th " Died. Autopsy.
113	J. H. P. 53 yrs. M.	Cheever.	Same day.	Knocked down on street by a horse.	Scalp wound, lft. frontal region, 2 inches. Bone exposed. Scalp wound, rt. frontal, 1 inch. Bone exposed.	Unconscious in 40 minutes after accident. Hemorrhage from nose and mouth.	None.	. . .	. . .	1 hour " Autopsy.
114	H. B. 44 yrs. M.	Gavin.	"	Struck by piece of wood from circular saw.	Scalp wound, 4 inches, rt. frontal region. Bone exposed.	None.	"	. . .	. . .	5th day. Rel'd.
115	J. M. E. 39 yrs. M.	Monks.	"	Struck by officer's club.	Vertex scalp wound 3 inches. Bone exposed.	"	"	. . .	. . .	9th " "
116	J. M. A. 28 yrs. M.	Monks.	One day.	Fell from moving train.	2 scalp wounds, lft. parietal region. Bone exposed.	"	"	. . .	. . .	3d " "
117	M. M. 35 yrs. F.	Monks.	Same day.	Knocked down by team.	Scalp wound, lft. parietal region. Bone exposed.	"	"	. . .	. . .	3d " "
118	E. D. F. 26 yrs. M.	Cheever.	"	Thrown from wagon.	Scalp wound, lft. parietal region. Bone exposed.	"	"	. . .	. . .	12th " "
119	M. R. 53 yrs. M.	Cheever.	"	Knocked down by a team.	Scalp wound, occiput, 3 inches. Bone exposed.	Delirious for several days.	Present.	10th. 3.84%.	Cont'd till disch.	12th " "
120	A. O. C. 26 yrs. M.	Cheever.	"	Fell from wagon.	Scalp wound, lft. parietal region. Bone exposed.	None.	None.	. . .	. . .	2d " "
121	F. D. S. 16 yrs. M.	Cheever.	"	Fell from wagon.	Scalp wound, lft. parietal region. Bone exposed. Several inches.	"	"	. . .	. . .	21st " "
122	G. A. 17 yrs. M.	Burrell.	"	Struck by locomotive.	Scalp wound, occiput, 2½ inches. Bone exposed. Compound fracture arm and leg.	Unconscious 24 hours. Delirious 4 days.	Present.	Trace after 12th.	3d day.	Still in hospital.
123	L. F. K. 46 yrs. M.	Monks.	"	Struck by club.	Scalp wound, lft. parietal, 1½ inches. Bone exposed.	Unconscious 24 hours. Delirious 4 days.	"	. . .	. . .	3d day. Rel'd.
124	C. A. P. 52 yrs. M.	Monks.	"	Kicked by horse.	Scalp wound, lft. frontal region, 2 inches. Bone exposed and chipped.	None.	None.	. . .	. . .	11th " "
125	E. McC. 40 yrs. M.	Cushing.	"	Clubbed on head.	Scalp wound, vertex. Bone exposed.	Delirious.	"	. . .	. . .	Still in hospital.
126	J. G. 27 yrs. M.	Bolles.	"	Struck by an officer's club.	Scalp wound, 3 inches, lft. frontal region. Bone exposed.	None.	"	. . .	. . .	6th day. Rel'd.
127	D. McG. 36 yrs. M.	Burrell.	"	Struck by a locomotive.	Scalp wound, lft. frontal and parietal region, 5 inches. Bone exposed.	"	"	. . .	. . .	Still in hospital.

were removed. The wounds healed almost throughout by primary union. On the sixth day a right facial paralysis was first noticed, with mouth drawn to the left, probably due to injury of the facial nerves. At night on the same day patient had difficulty in swallowing, and on the next day developed a pronounced tetanus in the facial muscles, with spasm of the tongue. Following this he failed rapidly and died on the eighth day.

Autopsy. — Brain normal, with dura adherent to the bone at the base. No fracture about the head except in the bones of the face. Right kidney was ruptured, with hæmorrhage into the suprarenal capsule. There was also hæmorrhage into the capsule of the liver, on right lobe under surface. There was an acute splenic tumor, congestion of lungs, and a few small patches of sclerosis in aorta and coronary arteries.

Urine. — An examination of the urine passed about twelve hours after the accident disclosed the presence of 1.04% of sugar, with a specific gravity of 1020, and a large trace of albumen. Repeated daily examinations of the urine gave a smaller percentage of sugar each day up to the sixth, when it had entirely disappeared. A positive test by fermentation.

As on the sixth day the first symptoms of tetanus appeared, it is perfectly evident that the glycosuria could not have been caused by this complication. To which of the other lesions it was due it is impossible to determine.

Case 112, Class II. — J. M., male, aet. 50. Service of Dr. Monks. Five days before, fell in getting off a moving electric car, striking head on a post. At time of entrance, on patient's head were two irregular suppurating cavities in scalp, the first over vertex about one by one and a half inches; the second over left parietal eminence, about one-half by two inches, appearing as if the flesh had been torn away, leaving the bone uninjured and exposed beneath. In six days erysipelas of the face and scalp developed, following a chill, headache, and a rise in temperature the day before. Patient did well until the twenty-second day, when without apparent cause he suddenly became unconscious and died.

Autopsy. — There was no evidence of fracture of the skull; great excess of serous fluid in the subarachnoid spaces and in the ventricles. The veins of the brain were distended with dark fluid blood. The puncta cruata were prominent; no meningitis. Brain substance firm, and on section no evidences of hæmorrhage or other pathological changes. Other organs of the body normal except for a moderate œdema and congestion of the lungs.

Urine. — The urine of this patient, from the time of entrance till his death, was examined frequently, and at all times was found to be above 1030 in specific gravity and to contain a large amount of sugar, the greatest amount at any one time being 5.55%, varying between that and 4%. There was active fermentation at each examination.

As the erysipelas developed on the sixth day, it may at once be excluded in considering the cause of the glycosuria. It is possible, in view of the early presence of a large quantity of sugar in the urine, continuing undiminished several weeks till death, that we have to do in this case with a diabetes existing prior to the injury. The previous history of the patient with respect to thirst, frequent micturition, etc., is, however, entirely negative. Charcot says that cases of persistent glycosuria following blows on the occiput are more common than, and are apt to be mistaken for, cases of true diabetes. It seems probable, then, that this may be a case of that kind.

Case 119, Class II. — M. R., male, aet. 53. Service of Dr. Cheever. Knocked down by a team in the street, causing over occiput an irregular lacerated wound three inches long, bone exposed. In addition, there was a fracture of one rib. Patient was very restless, with a mild delirium at times, for several days. The wound united well by primary union, and on the twelfth day he was removed from the hospital.

Urine. — The first urine examined was passed ten hours after the injury. At that time the specific gravity was 1033, with a trace of albumen and 3.84% of sugar. Two days later there was 4% of sugar present, with a specific gravity of 1043. From this time on until the patient's discharge the sugar gradually decreased to 1.91% at the last

No.	Name. Age. Sex.	Service.	Day of Injury before Entrance.	Cause of Injury.	Visible Lesion.	Symptoms.	Urine.			Day of Discharge. Result.
							Sugar.	Day of first appearance after injury.	Day of disappearance after injury.	
153	W. C. 56 yrs. M.	Bolles.	Same day.	Fell downstairs.	No external evidences of injury.	Unconscious for 2 hours; stupid for 48 hours.	None.	. . .	. . .	8th day. Rel'd.
154	C. H. S. 65 yrs. M.	Bradford.	"	Fell on icy pavement.	Contusion of head; no wound.	Unconscious about 1 hour; pupils contracted, not reacting.	"	. . .	. . .	3d day. Rel'd.
155	M. A. C. 45 yrs. F.	Bolles.	"	Fell down 4 steps.	Scalp wound 2 inches long over lft. frontal bone.	Unconscious about 1 hour.	"	. . .	. . .	9th day. Rel'd.
156	S. J. W. 63 yrs. M.	Bolles.	"	Thrown from carriage.	Hæmatoma over rt. parietal eminence.	Semi-conscious for short time.	"	. . .	. . .	2d day. Rel'd.
157	H. L. J. 43 yrs. M.	Bolles.	"	Thrown from carriage.	No external evidences of injury.	Semi-conscious for a short time.	"	. . .	. . .	2d day. Rel'd.
158	T. M. C. 14 yrs. M.	Bradford.	"	Fell 7 feet.	Hæmatoma over rt. parietal eminence.	Unconscious about 3 hours.	"	. . .	. . .	6th day. Rel'd.
159	J. W. 45 yrs. M.	Burrell.	"	Fell from electric car.	Scalp wound 1 inch long over rt. frontal eminence.	Unconscious about 2 hours; slight hemorrhage from nose.	"	. . .	. . .	11th day. Reld.
160	B. A. D. 27 yrs. F.	Burrell.	"	Struck by runaway horse.	Small scalp wound over vertex.	Semi-conscious for 2 days.	"	. . .	. . .	36th day. Reld.
161	M. D. 16 yrs. F.	Burrell.	"	Knocked down on street.	No external evidences of injury.	Unconscious about 1½ hours.	"	. . .	. . .	5th day. Reld.
162	J. H. D. 25 yrs. M.	Watson.	"	Fell on sidewalk.	Hæmatoma over lft. temporal bone.	Unconscious about 1 hour.	"	. . .	. . .	5th day. Rel'd.
163	T. C. R. 65 yrs. M.	Watson.	"	Fell downstairs.	Scalp wound ½ inch long over rt. frontal eminence.	Unconscious for 6 hours; Rt. pupil dilated.	"	. . .	. . .	5th day. Reld.
164	S. M. S. 45 yrs. F.	Watson.	"	Fell downstairs.	Scalp wound ¾ inch long over rt. frontal eminence.	Semi-conscious for 24 hours.	"	. . .	. .	11th day. Rel'd.
165	L. H. 45 yrs. F.	Watson.	"	Thrown from carriage.	Scalp wound 1 inch long over rt. occiput.	Semi-conscious for short time.	"	. . .	. .	3d day. Rel'd.
166	E. M. 54 yrs. M.	Bolles.	"	Fell 10 feet into elevator well.	Contusion on head.	Unconscious about 1 hour.	"	. . .	.	9th day. Rel'd.
167	S. J. O'C. 24 yrs. M.	Bolles.	"	Fell on sidewalk.	Contusion on head.	Unconscious for several hours; slight hemorrhage from left ear.	"	. .	. .	15th day. Rel'd.

Class III. — Concussion, Simple and with Scalp Wounds.

No.	Name. Age. Sex.	Service.	Day of Injury before Entrance.	Causes of Injury.	Visible Lesion.	Symptoms.	Urine.			Day of Discharge. Result.
							Sugar.	Day of first appearance aft'r injury.	Day of its disappearance aft'r injury.	
128	F. M. Q. 14 yrs. M.	Post.	Same day.	Fell 10 feet from ladder.	Scalp wound 2 inches long, occipital region.	Semi-conscious for 12 hours; some vomiting.	None.	. . .	. . .	4th day. Rel'd.
129	C. M. 59 yrs. M.	Post.	One day.	Fell on sidewalk, striking head.	Hæmatoma and contusion, rt. temporal region.	Semi-conscious for 24 hours.	"	. . .	. . .	8th day. Rel'd.
130	P. K. 22 yrs. M.	Smith.	Same day.	Fell from roof, two stories.	No external evidences of injury.	Unconscious (to death).	"	. . .	. . .	3d day. Died.
131	J. M. C. 43 yrs. M.	Post.	"	Fell 26 feet from building.	Scalp wound over rt. occipital bone.	Unconscious about 1 hour.	"	. . .	. . .	24th day. Rel'd.
132	N. F. 32 yrs. M.	Gavin.	Night before (about 12).	Assaulted; struck on head.	Small scalp wound over rt. parietal bone.	Semi-conscious about 12 hours.	"	. . .	. . .	7th day. Rel'd.
133	J. C. 5½ yrs. M.	Gavin.	Same day.	Fell from fence, striking head.	Hæmatoma rt. parietal bone.	Semi-conscious about 12 hours.	"	. . .	. . .	7th day. Rel'd.
134	M. B. 5 yrs. F.	Gavin.	"	Fell down two flights of stairs.	No external evidences of injury.	Unconscious about 12 hours; pupils dilated, and do not react.	"	. . .	. . .	8th day. Rel'd.
135	J. K. 10 yrs. M.	Gavin.	"	Fell on sidewalk, striking head.	No external evidences of injury.	Semi-conscious about 6 hours.	"	. . .	. . .	3d day. Rel'd.
136	A. C. 21 yrs. F.	Monks.	"	Fell on sidewalk, striking head.	Scalp wound 2 inches long over lft. parietal bone.	Unconscious about 1 hour.	"	. . .	. . .	5th day. Rel'd
137	P. McG. 55 yrs. M.	Monks.	"	Fell, striking head on curb-stone.	Scalp wound 1½ inches long over lft. parietal bone.	Unconscious about 10 hours.	"	. . .	. . .	2d day. Rel'd
138	W. F. 15 yrs. M.	Watson.	"	Fell 6 feet, striking head.	No external evidences of injury.	Semi conscious for a few hours.	"	. . .	. . .	3d day. Rel'd.
139	A. W. 64 yrs. F.	Cheever.	"	Fell downstairs.	Hæmatoma over lft. occipital bone.	Semi-conscious for a short time.	"	. . .	. . .	30th day. Rel'd,
140	J. E. D. 22 yrs. M.	Gavin.	Two days.	Unknown.	Small lacerated wounds over lft. parietal and frontal bones.	Semi-conscious for 3 days; Ptosis left eyelid.	"	. . .	. . .	15th day. Rel'd.
141	W. K. 32 yrs. M.	Monks.	Same day.	Bale cotton fell 10 feet, striking head.	No external evidences of injury.	Unconscious about 12 hours; delirious later.	"	. . .	. . .	3d day. Rel'd.
142	J. F. 24 yrs. M.	Monks.	"	Unknown.	No external evidences of injury except contusion about left eye.	Unconscious about 12 hours; hemorrhage from left ear; left pupil dilated.	"	. . .	. . .	3d day. Rel'd.
143	J. O. F. 24 yrs. M.	Cheever.	"	Fell 30 feet from staging.	No external evidences of injury.	Unconscious a few hours; left pupil dilated; does not react.	"	. . .	. . .	5th day. Rel'd.
144	P. P. K. 7 yrs. M.	Gavin.	"	Kicked on head.	Large contused area rt. parietal region.	Semi-conscious about 15 hours.	"	. . .	. . .	15th day. Rel'd.
145	F. E. 12 yrs. M.	Gavin.	"	Fell from wall, 10 feet.	No exteroal evidences of injury.	Unconscious for a short time.	"	. . .	. . .	9th day. Rel'd.
146	J. H. F. 22 yrs. M.	Cheever.	"	Fell downstairs.	No external evidences of injury.	Unconscious about 60 hours; mental symptoms following.	"	. . .	. . .	30th day. Rel'd.
147	J. S. 12 yrs. M.	Gavin.	"	Thrown from carriage.	Hæmatoma over rt. parietal bone.	Unconscious about 2 hours.	Present.	2d day.	9th day.	12th day. Well.
148	D. H. 28 yrs. M.	Watson.	"	Fell on sidewalk.	Hæmatoma over occiput.	Slight concussion.	None.	. . .	. . .	3d day. Rel'd.
149	M. C. 37 yrs. F.	Watson.	Seven days.	Fell on ice.	Scalp wound over lft. frontal bone.	Unconscious about 15 hours.	"	. . .	. . .	28th day. Well.
150	P. I. 23 yrs. M.	Bradford.	Two days.	Struck on back of head.	Hæmatoma over occiput.	Unconscious about 5 days.	"	. . .	. . .	17th day. Well.
151	W. H. N. 42 yrs. M.	Bolles	Same day.	Struck on head by descending elevator.	No external evidences of injury.	Unconscious about 12 hours; convulsions; paralysis right arm; sub-conjunctival hemorrhages.	"	. . .	. . .	18th day. Rel'd.
152	D. F. 45 yrs. M.	Bolles.	"	Struck by falling hatch of vessel.	Scalp wound over vertex.	Unconscious from 1 to 2 hours.	"	. . .	. . .	6th day. Rel'd.

examination on the twelfth day. There was a pronounced yeast fermentation at all times.

Case 122, Class II. — G. A., male, aet. 11. Service of Dr. Burrell. While crossing the railroad track was struck by a locomotive, causing a large hæmatoma over occiput, with a scalp wound two and a half inches long, bone exposed beneath. In addition, there was a compound fracture of the right leg and a bad crush of right arm, necessitating amputation above the elbow. The boy suffered from profound shock, remaining unconscious for about twenty-four hours, followed by delirium lasting four to five days. He is now making an excellent recovery, still remaining in the hospital.

Urine. — The first urine was passed twelve hours after the injury. It contained a trace of albumen, a very small percentage of sugar, and had a specific gravity of 1028. On the third day the trace of sugar had disappeared and frequent examinations up to the present time have not detected its presence again.

Class III.

Case 147, Class III. — J. S., male, aet. 12. Service of Dr. Gavin. Was thrown from a carriage in a "runaway" accident, striking on his head. He was picked up unconscious, remaining in this condition for about two hours. On arrival at the hospital there was found a hæmatoma over the right parietal and occipital regions. No depression or fracture of the bones could be made out. Pupils were equal and reacted to light. He complained of slight pain in the head, this, however, being the only subjective symptom. The patient was discharged relieved on the eleventh day, in a very good general condition.

Urine. — The urine was unfortunately not examined for thirty-six hours, at which time 1.2% of sugar was found. On the fourth day after injury it was found that the sugar in the urine had diminished to 0.3% ; on the sixth day only a faint trace, and a complete disappearance on and after the ninth day. At each examination of the urine the fermentation test with yeast was tried with a satisfactory reaction, as long as the sugar remained. At no time were we able to get a reaction for acetone or diabetic acid. On the thirty-

seventh day after the injury another specimen of urine was obtained from the patient, and was found to be free from sugar.

Class IV.

Case 169, Class IV. — W. M., male, aet. 40. Service of Dr. Gavin. Shot himself in the head during a fit of melancholia. The patient was brought to the hospital in a semi-conscious condition, having a perforated wound one and a half inches above and same distance back of the right ear. The pupils were equal and reacted to light, no paralysis. The wound was probed by Dr. Gavin, who found an opening having jagged edges through the skull. An effort to find the bullet was unsuccessful. Two days later the patient regained consciousness, and at this time slight paralysis of the left side, with deviation of the eyes to the right, was noticed. On the fifth day became delirious and noisy, this continuing for four days. There was retention of urine on the tenth day, and at this time there was a sero-purulent discharge from the right ear. The patient's mental condition had improved somewhat and the general condition remained pretty good, to the twenty-eighth day, when he became unconscious; the retention of urine had given way to incontinence, and still paralysis of left side. Death occurred on the twenty-ninth day.

Autopsy. — A well-marked cicatrix situated two inches behind and one and one-half inches above external angular process. On removing the scalp a circular opening through skull was found. A fracture one-half inch behind coronal suture, penetrating the great wing of the sphenoid bone. Bullet was found just inside the opening in the skull and over the third frontal convolution just anterior to the ascending limb of Sylvius. This frontal convolution was softened and destroyed. There was no abscess cavity at this point.

Urine. — An examination of the urine made seventeen hours after the injury had a sp. gr. of 1025; albumen, slightest possible trace; sugar, a trace, too slight to be quantitated. Repeated examinations were made during the life of the patient, but no sugar was found at any time after the first examination.

No.	Name. Age. Sex.	Service.	Day of Injury before Entrance.	Cause of Injury.	Visible Lesion.	Symptoms.	Urine.			Day of Discharge. Result.
							Sugar.	Day of first appearance after injury.	Day of disappearance after injury.	
168	T. O'B. 34 yrs. M.	Post.	Same day.	Struck on head by iron bar.	Scalp wound 2 inches long over lft. parietal bone. Bone depressed.	None.	None.	. . .	. . .	18th day. Rel'd.
169	W. M. 40 yrs. M.	Gavin.	"	Shot in head.	Compound fracture rt. parietal bone.	Unconscious about 12 hours. Paralysis lft. arm on second day. Deviation of eyes to rt.	Present.	17 hrs.	Within 24 hrs.	23th " Died. Autopsy.
170	T. M. McK. 10 yrs. M.	Gavin.	"	Struck on head by piece of wood.	Scalp wound 1½ inches long over lft. parietal bone. Bone depressed	Unconscious about 12 hours.	None.	. . .	. . .	15th day. Rel'd.
171	B. B. 2 yrs. F.	Gavin.	"	Fell from second-story window.	Scalp wound over rt. parietal bone. Fracture extending toward base	None.	"	. . .	. . .	15th " "
172	H. H. 37 yrs. F.	Watson.	Two days.	Shot in head.	Perforating wound ½ inch above external angular process.	"	"	. . .	. . .	24th " "
173	T. O'N. 24 yrs. M.	Watson.	Same day.	Struck on head by a brick.	Hæmatoma 1½ inches from median line over rt. frontal bone. Slight laceration of scalp. Depressed bone.	Unconscious about 12 hours. Left pupil dilated, and does not react. Paralysis of rt. side.	"	. . .	. . .	27th " "
174	M. D. 37 yrs. M.	Gavin.	"	Struck on head by lump of coal.	Small scalp wound over vertex. Bone depressed.	None.	"	. . .	. . .	14th " "
175	R. B. 45 yrs. M.	Gavin.	"	Fell from second-story window.	Scalp wound 1 inch long over lft. frontal bone. Bone depressed and comminuted. Fragments of bone removed and part of roof of orbit.	Unconscious (to death). Delirium at times.	"	. . .	. . .	7th " Died.
176	J. P. M. 60 yrs. M.	Gavin.	"	Fell from ladder, 60 feet.	Scalp wound 2 inches long over lft. parietal, and another 3 inches long over frontal bone, median line.	Unconscious (to death).	"	. . .	. . .	7th " "
177	L. K. 13 yrs. M.	Gavin.	"	Struck on head by block of wood.	Scalp wound 2 inches long over rt. frontal bone. Bone depressed	None.	"	. . .	. . .	8th " Rel'd.
178	E. W. 40 yrs. F.	Cheever.	"	Shot in head.	Perforating wound ½ inch from outer canthus. Fracture floor orbit and malar bone. Wound through antrum.	Hemorrhage from rt. nostril.	"	. . .	. . .	45th " "
179	L. M. 37 yrs. M.	Bradford.	"	Struck on head with beer bottle.	Scalp wound over rt. frontal bone and fracture of outer table.	None.	"	. . .	. . .	14th " "
180	M. K. 52 yrs. M.	Bolles.	"	Struck on head with ox-goad.	Scalp wound over rt. frontal eminence, and fracture.	Stupor for 3 days. Subconjunctival ecchymosis both eyes.	Present.	2d day.	Cont'd. till disch.	19th " "
181	F. R. 19 yrs. M.	Bolles.	"	Struck with an iron bar.	Scalp wound over rt. temporal bone. Fracture with depression of bone.	None.	"	9th day.	11th day.	19th " "
182	J. T. 15 yrs. M.	Watson.	Seven days.	Thrown against bar iron.	Scalp wound over rt. frontal bone. Fracture and depression of bone	"	None.	. . .	. . .	47th " Well.
183	J. R. 24 yrs. M.	Bolles.	Same day.	Struck on head with heavy piece of coal.	Scalp wound over vertex. Depressed fracture.	Unconscious (to death). General spastic condition.	Present.	8 hrs.	Not exam. again.	12 hrs. Died.
184	M. L. F. 36 yrs. M.	Watson.	"	Struck with "monkey-wrench."	Scalp wound and fracture of lft. frontal bone.	None.	None.	. . .	. . .	15th day. Rel'd.
185	H. F. W. 15 yrs. M.	Bradford.	"	Struck over head with sabre.	Scalp wound 2 inches long over vertex. Fracture outer table.	"	"	. . .	. . .	3d " "
186	J. S. 39 yrs. M.	Bolles.	"	Struck with hatchet.	Two scalp wounds over lft. frontal eminence. Fracture of outer table.	"	"	. . .	. . .	13th " "

No.	Name. Age. Sex.	Service.	Day of Injury before Entrance.	Cause of Injury.	Visible Lesion.	Symptoms.	Urine.			Day of Discharge. Result.
							Sugar.	Day of first appearance after injury.	Day of Disappearance after injury.	
187	B. C. 38 yrs. M.	Bradford.	Same day.	Load of coal fell on him.	Several scalp wounds over occiput. Bone depressed.	None.	None.	. . .	. . .	24th day. Rel'd.
188	J. A. C. 33 yrs. M.	Burrell.	"	Struck with block of wood.	Scalp wound 1½ inches long between eyebrows. Depressed outer table.	Semi-conscious for ½ hour.	"	. . .	. . .	8th " "
189	E. S. 36 yrs. M.	Post.	Five days.	Struck with falling plank.	No external evidences of injury. Depressed bone felt over occiput.	Unconscious for ½ hour, and again on third day for 36 hours, with convulsions and vomiting.	"	. . .	. . .	13th " "
190	J. D. 25 yrs. M.	Watson.	One day.	History of assault, means unknown.	No external evidences of injury.	Unconscious for 24 hours, then delirious. Reflexes intact. Paralysis of left rectus.	"	. . .	. . .	60th " Died. Autopsy.
191	G. M. R. 14 yrs. M.	Watson.	Same day.	Struck by a shaft.	Compound fracture of frontal and nasal bones, including fracture floor of lt. orbit.	None.	Present.	40 hrs.	Only exam. once.	10th day. Rel'd.

Case 191, Class IV. — G. M. R., male, aet. 14. Service of Dr. Watson. Was struck on the head by the shaft of a planing machine, sustaining a lacerated wound extending from bridge of nose to supraorbital ridge of frontal, destroying left eye, and involving the nasal, frontal, malar, and possibly superior maxillary bones in compound comminuted fracture. On entrance to the hospital was conscious, pulse rapid, but of low tension. The patient was operated upon by Dr. Monks, who enucleated all remains of the eye, removed fragments of bone, thoroughly cleansed and dressed the wound antiseptically. The next day, while dressing the wound, a rather profuse discharge of sero-sanguinous matter was observed. There followed a good recovery, and he was discharged on the nineteenth day.

Urine. — An examination of the urine twenty-four hours after the injury showed sp. gr. 1035, slightest possible trace of albumen, 0.85% of sugar. The fermentation test was active. During the patient's stay in the hospital unfortunately the urine was not examined again, but five months after the injury the patient returned for observation, and the urine was found to have a sp. gr. of 1020, slight trace of albumen, and contained no sugar.

Case 180, Class IV. — M. K., female, act. 52. Service of Dr. Bolles. In an encounter with a burglar was struck twice on the head with an unknown implement. She remained in an unconscious condition about four and half hours. On entrance, the patient was found to be a fleshy woman with a scalp wound about three inches in length over the right frontal eminence and a crack in the bone running forward. No depression of bone. The wound was treated antiseptically. No preëxisting history or symptoms of diabetes in patient or in ancestors. An uninterrupted recovery followed, without any notable subjective symptoms. The patient was discharged on the ninteenth day.

Urine. — *Eighteen hours* after entrance, sp. gr. 1040; albumen, very slight trace; sugar present (not quantitated.) The amount of urine passed in the first twenty-four hours was 1,080 c.c.

Third day. Amt., 960 c.c. Sp. gr. 1037, and albumen very slight trace; sugar, 2.5%. Marked fermentative reaction.

Fourth day. Amt., 1,260 c.c. Sp. gr. 1024; albumen as before; sugar, 1.19%. Marked fermentative reaction.

Seventh day. Amt., 1,500 c.c. Sp. gr. 1016: albumen as before; sugar, 0.35%. Very slight fermentation.

Ninth day. Amt., 1,800 c.c. Sp. gr. 1016; albumen as before. Sugar present, but too slight to quantitate. No fermentative reaction.

Eleventh day. Amt., 1,380 c.c. Sp. gr. 1028; albumen, a slight trace; sugar, a trace, too slight to quantitate.

Twelfth day. Amt., 1,830 c.c. Sp. gr. 1030; albumen, a slight trace; sugar, 3.84%. Fermentation very active.

Thirteenth day. Amt., 1,260 c.c. Sp. gr. 1042; albumen as before; sugar, 4.16%. Fermentation very active.

Fifteenth day. Amt., 650 c.c. Sp. gr. 1042; albumen as before; sugar, 5.43%. Fermentation very active.

Sixteenth day. Amt. accidentally lost. Sp. gr. 1036; albumen as before; sugar, 2.38%. Fermentation active.

Nineteenth day. Amt., 1,350 c.c. Sp. gr. 1033; albumen, very slight trace; sugar, 1.29%. Fermentation active. Discharged on this day.

Acetone and diabetic acid were not obtained at any time, although tested for at each examination of the urine.

There are some points in this case which would tend to show a preëxisting permanent diabetes; viz., a corpulent individual, the continued presence and invariable large percentage of sugar. On the other hand, we could get no history of diabetes (pruritus, excessive thirst, a noticeable increase in the daily amount of urine, etc.). The daily amount of urine, while in the hospital, was generally below 1,500 c.c. (although this might well be in a case of permanent diabetes).

Case 181, Class IV. — F. R., male, aet. 19. Service of Dr. Bolles. During a quarrel he was struck on the head with an iron bar about one inch in diameter. His mind was perfectly clear after the accident. On entering the hospital there was found a scalp wound about three inches in length over the right temporal bone, with an exposure and depression of bone. The depression extended over an area of about half an inch and about one-eighth inch in depth. There were no subjective symptoms except for rather severe head-

Class V. — Fractures of Base of Skull.

No.	Name. Age. Sex.	Service.	Day of Injury before Entrance.	Cause of Injury.	Visible Lesion.	Symptoms.	Urine.			Day of Discharge. Result.
							Sugar.	Day of first appearance after injury.	Day of disappearance after injury.	
192	M. S. 40 yrs. M.	Watson.	Same day.	Assaulted, struck on head.	No external evidences of injury.	Semi-conscious for a few hours and then unconscious (to death). Hemorrhage from lft. ear and nose.	None.	. . .	. . .	5th day. Died. Autopsy.
193	C. H. C. 54 yrs. M.	Monks.	"	Struck on head by piece of iron.	Hæmatoma over rt. parietal and frontal bones. Bone comminuted.	Unconscious (to death). Pupils do not react. External strabismus.	Present.	6 hrs. 2.17%.	. . .	9 hrs. Died.
194	A. M. 3 yrs. M.	Gavin.	"	Knocked down and run over by team.	Contusion of left side of face and head.	Semi-conscious for 36 hours. Hemorrhage from rt. ear.	None.	. . .	. . .	24th day. Rel'd.
195	M. K. 47 yrs. F.	Monks.	"	Fell down cellar stairs.	No external evidences of injury.	Unconscious (to death).	"	. . .	. . .	4th " Died.
196	T. O'C. 27 yrs. M.	Monks.	One day.	Assaulted, means unknown.	No external evidences of injury.	Unconscious (to death). Hemorrhage from lft. ear.	Present.	20th. 1%.	. . .	30 hrs. " Autopsy.
197	M. M. 16 yrs. M.	Cheever.	Same day.	Fell from wagon.	Scalp wound 1½ inches long over rt. temporal bone.	Hemorrhage from lft. ear, followed by serous discharge and deafness.	None.	. . .	. . .	16th day. Rel'd.
198	J. J. G. 29 yrs. M.	Cheever.	"	Fell from ladder.	No external evidences of injury.	Unconscious for 8 days. Hemorrhage from rt. ear.	"	. . .	. . .	15th " "
199	J. E. F. 20 yrs. M.	Cheever.	"	Fell 30 feet.	Several small scalp wounds over lft. parietal bone.	Unconscious for 24 hours. Profuse hemorrhage from rt. ear.	"	. .	. . .	10th " "
200	C. M. M. 70 yrs. M.	Bolles.	"	Fell on sidewalk, striking head.	Scalp wound over lambdoid suture.	Unconscious for 6 hours, and again after fifth day (to death). Conjugate deviation of eyes. Left pupil dilated not reacting. Hemorrhage from lft. ear followed by serous discharge.	"	. . .	. . .	7th " Died.
201	J. S. 37 yrs. M.	Bolles.	One day.	Fell into hold of vessel.	Scalp wound 3 inches long over occipital bone. Fracture extending to base.	Unconscious (to death). Hemorrhage from rt. ear.	Present.	24 hrs. 1.25%.	. . .	34 hrs. "
202	J. P. D. 50 yrs. M.	Bolles.	Same day.	Fell from electric car.	Hæmatoma of scalp.	Hemorrhage from lft. ear and slight from mouth. Unconscious ½ hour. Stupor for 48 hours.	None.	. . .	. . .	12th day. Rel'd.
203	J. L. 28 yrs. M.	Bolles.	"	Struck with hammer.	No external evidences of injury.	Hemorrhage from lft. ear. Subconjunctival hemorrhage of lft. eye. Unconscious 6 hours. Aphasia.	"	. . .	. . .	9th " "
204	J. F. 35 yrs. M.	Bradford. Watson.	"	Struck by a locomotive.	Scalp wound over left occiput.	Unconscious for 36 hours. Paralysis of rt. face, arm, and leg. Aphasia.	"	. . .	. . .	68th " "
205	J. S. 45 yrs. M.	Watson.	"	Unknown.	Wound of jaw bone exposed.	Unconscious for ½ hour. Hemorrhage from lft. ear.	"	. . .	. . .	8th " "
206	T. F. M. 33 yrs. M.	Bolles.	"	Fell 8 feet, striking on head.	No external evidences of injury.	Unconscious for 2 hours. Hemorrhage from ears and nose. Severe headache.	"	. . .	. . .	12th " "
207	W. D. 70 yrs. M.	Bradford.	"	Knocked down on street.	Lacerated wounds of forehead.	Hemorrhage from lft. ear. Delirium 2 days later.	"	. . .	. . .	7th " Died.
208	G. L. 23 yrs. M.	Bradford.	"	Fell 20 feet on to frozen ground.	No exteroal wound.	Unconscious 6 days. Frequent vomiting. Paralysis of rt. arm and leg.	Present.	3d day.	. . .	29th " Rel'd.
209	G. B. 32 yrs. M.	Bradford.	"	Fell in elevator well, 4 stories.	No external wound. Comp. fracture of jaw.	Unconscious for several hours, followed by delirium for 10 days. Hemorrhage from both ears and nostrils, followed by serous discharge from ears.	None.	. . .	. . .	30th " "
210	D. G. 26 yrs. M.	Bolles. Watson.	"	Fell 10 feet on stone steps.	Scalp wound over vertex.	Stertorous resp. Pulse slow. Unconscious (to death).	"	. . .	. . .	20 hrs. Died.
211	E. M. 48 yrs. M.	Watson.	"	Struck by descending elevator.	Scalp wound 2 inches long over rt. parietal eminence.	Hemorrhage from rt. ear.	"	. . .	. . .	12th day. Rel'd.
212	C. A. L. 42 yrs. M.	Watson.	"	Thrown from carriage.	Large hematoma over rt. parietal eminence.	Unconscious (to death). Hemorrhage from lft. ear.	Present.	16 hrs.	not exam. again.	30 hrs. Died.

ache. The temporal bone was trephined and several small pieces of bone removed. No subjective symptoms. A good recovery followed, and he was discharged on the nineteenth day.

Urine. — At the first examination made three days after injury, there was a sp. gr. of 1028, and no sugar or albumen present. On the sixth day the urine had a sp. gr. of 1030 and a very slight trace of albumen and sugar. There was a complete disappearance of the sugar two days later, and no recurrence during his stay in the hospital.

Case 183, Class IV. — J. R., male, aet. 24. Service of Dr. Bolles. Was struck on the head by a large piece of coal which fell from a staging above. He was knocked down and was insensible for only a few moments. He then walked home without assistance. Three hours and a half later he became unconscious and was brought to the hospital in this condition. Examination showed a well developed man, pulse rapid (160), resp. 40, and simulating the Cheyne-Stokes character. Both pupils dilated — left more than right — and reacting sluggishly to light. A small scalp wound at the coronal suture, in the median line. All extremities were rigid, relaxing momentarily at times. The operation of trephining was performed by Dr. Bolles, who removed a button at the point of scalp wound and another one over the course of the left middle meningeal artery, but nothing was found in either place to account for the symptoms. Death occurred fifteen hours after the injury. There was no autopsy.

Urine. — About eight hours after the injury the urine was drawn by catheter, and found to have a sp. gr. of 1033; albumen, a very slight trace; sugar, a faint trace (too slight to quantitate). Only one examination was made before death.

Class V.

Case 212, Class V. — C. A. L., aet. 42, male. Service of Dr. Watson. Was thrown out of carriage one half hour before entrance. When seen first there was slight stupor, from which he could be aroused sufficiently to give name and age, but relapsed immediately on being allowed to remain

undisturbed, and at the end of another hour was totally unconscious, soon becoming delirious. Over right parietal region was a large swelling beneath which nothing could be felt; hæmorrhage from left ear; no paralysis; knee-jerks slightly increased; examination otherwise negative. About three hours after the accident an incision was made over swelling, revealing a crack in the bone, running downwards and forwards towards the base. Bone was trephined, finding the dura lacerated with considerable hæmorrhage, necessitating tying the posterior branch of the middle meningeal artery. Patient did not recover consciousness after the operation. He continued noisy and delirious, dying forty-five hours after the accident.

Urine. — It was possible to obtain only one specimen of urine from the patient. A small quantity was drawn by catheter sixteen hours after the injury. At that time it contained a very slight trace of albumen, with a sp. gr. of 1030, and a trace of sugar was found by Fehling's and the fermentation tests.

Case 201, Class V. — J. S., aet. 37, male. Service of Dr. Bolles. Twenty-four hours before entrance fell into the hold of a vessel striking on head. At entrance unconscious, very restless, and delirious; no paralysis. Over occiput a scalp wound three inches long, bone exposed beneath, in which a fracture was found extending into base beyond limits of wound. No depression or fissure. There was a bloody serous discharge from right ear, also a slight exophthalmos of right eye. Patient continued delirious and died thirty-four hours after the injury. No autopsy.

Urine. — Catheter specimen was obtained twenty hours after fall. Sp. gr. was 1027, containing a trace of albumen, and sugar to 1.25%. This was the only examination made.

Case 208, Class V. — G. L., aet. 23, male. Service of Dr. Bradford. Shortly before entrance fell twenty feet, striking on frozen ground. No external signs of violence; paralysis of right arm and leg. Unconscious, delirious, and very restless, lasting for about six days without other special subjective symptoms. Patient then made gradual improvement mentally, and in the use of right arm and leg, and on the twenty-ninth day, being up and walking about, was discharged.

Urine. — On the third day after the injury the urine was found to contain a very small amount of sugar with a slight trace of albumen, and a sp. gr. of 1030. This was the only time that sugar was found present.

Case 193, Class V. — C. H. C., aet. 54, male. Service of Dr. Monks. On the morning of entrance patient was struck on the head by a piece of falling machinery, was brought to the hospital unconscious, cyanotic; no paralysis; respiration slow and stertorous; pupils fixed, and there was slight external strabismus of both eyes. Over right parietal and temporal regions was a large hæmatoma, beneath which nothing could be distinguished. There was no hæmorrhage from ears, nose, or mouth. An immediate operation was performed, and on removing the flap, over an area two inches in diameter, the bone was found comminuted and depressed, with line of fracture extending into nose. Beneath, the dura was badly lacerated. Between the bone and dura was a large clot from the middle meningeal, which was ligated. Fragments of bone and the clot were removed, and the wound closed and drained. Patient failed rapidly, dying nine hours after the injury. No autopsy.

Urine. — The only specimen of urine obtained was drawn by catheter about six hours after the accident, immediately before operation. It had a sp. gr. of 1022, the slightest possible trace of albumen, and 2.17% of sugar.

Case 196, Class V. — T. O'C., aet. 27, male. Service of Dr. Monks. Twenty hours before entrance to hospital was assaulted in an unknown manner. Patient was unconscious, very restless and delirious, with no marks of injury about head. There was evidence of old hæmorrhage from left ear, but otherwise physical examination was negative. Death occurred about thirty hours after injury.

Autopsy. — Over left occipito-parietal region there was considerable infiltration of the scalp over an area four inches in diameter. Skull was fractured, beginning at about an inch to the left of the junction of the occipital and two parietal bones and extending to the left downwards and forwards across parietal and temporal bones, through the petrous, and stopping near the middle of squamous portion. There was no fissure or depression. Surface of brain vessels

markedly injected with little free blood within arachnoid spaces. At the base pia moist and opaque, covered with a thick exudation of pus and serum. To a less degree, some exudation found along sulci from base to vertex. Base of frontal lobes bruised over an area one inch in diameter and one-quarter inch deep. Also slight bruising at tip of left temporal lobe. Substance of brain moist and soft. Kidneys slightly enlarged, injected, and granular. Organs otherwise normal.

Anatomical Diagnosis. — Fracture base of skull, contusion of brain, and purulent meningitis.

Urine. — A catheter specimen was obtained immediately after entrance, twenty hours after the injury. The sp. gr. was 1030, it had a large trace of albumen, and sugar was present to less than one per cent., too small to determine quantitatively.

RESULTS.

Total number of cases examined	212	
" " " " glycosuria	20	9.43%
Class I.		
Number of cases	84	
" " glycosuria	5	5.95%
Class II.		
Number of cases	43	
" " glycosuria	4	9.3%
Class III.		
Number of cases	40	
" " glycosuria	1	2.5%
Class IV.		
Number of cases	24	
" " glycosuria	5	20.8%
Class V.		
Number of cases	21	
" " glycosuria	5	23.8%

These figures show the results obtained in percentages of cases having sugar, taken both collectively and as they have been arranged in classes.

Assuming that of all the twenty cases which had glycosuria none were diabetic before the injury, there is then a percentage of 9.43 in general for all classes of head injuries. This is fully as high as could be expected, considering the large proportion of the whole number of cases (167 out of 212) in Classes I., II., and III. These would generally be considered as minor accidents.

It does not seem unfair to argue upon the assumption that in all the patients the glycosuria occurred as the results of the trauma, and that they were not diabetic before. This seems especially reasonable when the fact is taken into consideration that of one thousand five hundred and sixty-three patients, the whole number of accident cases admitted to the hospital for the same thirteen months during which the head cases have been studied, no case apart from those with head injuries was found to have glycosuria. As it is, there are only three cases out of the whole number : J. M., Case 112, Class II. ; M. R., Case 119, Class II. ; and M. K., Case 180, Class IV., in which there can be any question. From a careful study of these after the accidents, and taking into consideration their previous histories, it seems very probable that a permanent glycosuria was consequent upon the head injury, rather than that a diabetes existed previously.

With the exception of Class III., which shows a surprisingly small percentage of sugar cases, there is a considerable increase in the number with each higher class. This is, as would naturally be expected, very noticeable in the increase of the last two over the preceding ones. The difference between these two considered together, apart from the other classes, is not sufficient to be of any marked significance. It has been of considerable interest, however, to consider them separately in this paper.

In general, then, it can be said, that all classes of head injuries taken together, about 10% manifest glycosuria as the result of traumatism, while the severe cases, fractures both of the vertex and base, from 20% to 25% have glycosuria.

Sugar may be present in the urine following any head injury, however slight. It is present much more frequently following severe injuries. From a diagnostic standpoint it is difficult to see how the presence of a glycosuria can render any assistance.

Injuries to the Spine and Back.

During the period over which our observations have extended, a limited number of cases of injury to the spine and back have been followed, but intentionally left out of the tables. According to Vogel (31), Warren (32), Ebstein (33), and others, transitory glycosuria, but more often permanent diabetes, may follow such injuries, and considered by most of the above observers to be due to concussion of the spine. Of such cases seen in the hospital during the thirteen months, none presented sugar in the urine, although carefully sought for.

Examination of the Urine.

The greatest care has been used in the examination of the urine of all cases, and in each especial reference made to the tests for sugar.

Fehling's solution was used for both the qualitative and quantitative tests. The routine qualitative tests were made as follows: Taking about 3 to 5 c.c. of a freshly prepared Fehling's solution, boiling and then adding 5 to 15 drops of the urine, from which the albumen (if more than a very slight trace) had been previously removed. The solution was *not* boiled after the addition of the urine, but immediately set aside and carefully watched for a reduction. If no reduction of the copper appeared within an hour or two, it was set aside for twenty-four hours and again examined for a deposit of the suboxide of copper. The amounts of sugar which have been designated as a *trace* and a *very slight trace*, appeared only after twenty-four hours, and in every instance a distinct precipitate of the suboxide of copper was found. No effort to quantitate slight traces of sugar was undertaken, for such would have been useless. In every case, however, where the amount of sugar was sufficient, a quantitative test was made.

In most of the sugar cases the fermentation test was made, not as a means of quantitation, but to detect the presence of glucose, and in all cases in which it was used, it proved confirmatory.

Albumen, together with casts and abnormal blood, was found in every case containing sugar, probably secondary in most instances, to the renal irritation produced by the sugar, even though it had been eliminated only a short time.

Since acetone and diabetic acid are a frequent accompaniment of sugar in the urine, especially in the permanent forms of diabetes, it is of interest to note that they were not found in any of the sugar cases here recorded. Ebstein (33) reports a case in whose urine acetone and diabetic acid were found not until eight years after an injury, resulting in a permanent diabetes. As far as found, there is no literature bearing on this subject, in those cases where sugar follows immediately or in a few days a head injury. It would appear, then, in cases J. M., Case 112, Class II.; M. R., Case 119, Class II.; and M. K., Case 180, Class IV., where there is some question as to a permanent preëxisting diabetes, that a point in favor of a permanent glycosuria following the injury is the absence of acetone and diabetic acid.

Age and Sex.

The ages of the cases where sugar was found varies between four and a half and fifty-four years, and are quite evenly distributed between these limits. It is therefore concluded from such cases as here observed that the age has no practical significance.

As regards the sex, very little of value can be drawn. Although 85% of the cases having sugar were males, yet it appears to be relative, much more, to the liability to injury (as among manual laborers) than to the sex alone.

Diet.

From the fact that the diet is oftentimes an important factor in the appearance and disappearance of sugar in the urine, it might be well to state, in a few words, that it had little or no attention in those cases were sugar was found. Following the injury the patient had milk in sufficient quan-

tities, until the general condition warranted a more generous and substantial diet of meat, vegetables, etc. It is therefore obvious that this question has practically no weight in the sugar cases considered.

Conclusions.

1st. That after injury sugar may appear in the urine as early as six hours and disappear within twenty-four, the average time for its appearance, however, being from eight to twelve hours; for the disappearance of the same, from the fifth to the ninth day.

2d. That a small proportion of the cases may exhibit a permanent glycosuria from the date of injury to the head.

3d. That acetone and diabetic acid are rarely if ever found in such cases, excepting where the condition becomes a permanent glycosuria, and even then probably only after a number of months or years.

4th. That of the twenty sugar cases here recorded, eleven (55%) had received an injury to the right side of the head, five (25%) to the left side, three (15%) to the occiput, and two (10%) where there was no external evidences of violence.

5th. That it is impossible in the present state of the knowledge of the pathology of diabetes and glycosuria to draw any inferences from the autopsies which have been obtained. It was thought best, however, to report them in full.

6th. That there is little to be said in regard to the mortality. Of the twenty cases eight died, six being the direct result of severe injuries, one from intercurrent disease and the third probably from alcoholism. In the two hundred and twelve cases sixteen were fatal, 50% of these having glycosuria.

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XX.

A CASE OF ACTINOMYCOSIS.

By F. B. Mallory, M.D.

Actinomycosis is a sufficiently rare disease in this part of the world to render a case coming to autopsy at the Boston City Hospital of enough importance to be reported. Unfortunately the nature of the disease was not recognized either clinically or at the post-mortem examination, so that many queries which were raised later could not be definitely settled owing to a lack of investigation at the proper time.

The patient, a native of Denmark, came from the West several months before his death. Further than that nothing is known of his previous history. He entered the Massachusetts General Hospital on the 2d of June, 1894. His temperature was then 99.1° F., his pulse 120, and his respirations 28. He complained of gradual failure of breath, and of the development of a cough. About two weeks before entrance he had noticed a lump in his epigastrium.

Physical Examination. — A weak, emaciated man with anxious expression. Heart-sounds heard loudly in chest, anemic murmur. Nothing found in lungs except dulness in upper left front. Liver dulness one-half inch below ribs. Skin on left epigastrium, over an area of several square inches, reddened and tense. Abdominal wall thickened all around and dull on percussion. Abscess opened and considerable foul material looking like broken-down tuberculous tissue escaped. Packed with iodoform gauze and poulticed. The wound was dressed daily, but remained thoroughly indolent, so that on June 14th the patient was etherized and the granulations curetted, the curette laying bare the cartilage of the left ribs and the abdominal fascia. No dead bone was found.

No further notes were made on the case, but the patient left the hospital soon after the first of July.

On the 23d of August he was admitted to the Boston City Hospital. He was then "dull in mind" and "talked very thick," so that his words could not be understood. No history concerning him could be obtained.

Physical Examination. — Poorly nourished and emaciated. Heart-sounds weak and rapid. Apex in mammary line, one and three-fourths inches below nipple. Râles and bronchial breathing over the apices of both lungs, front and back. Percussion subtympanitic on the left side. About one and one-half inches below xiphoid and running parallel with the costal border, is a wound about two inches long, which discharges foul-smelling, purulent, whitish fluid. Between the tenth and eleventh ribs, about three inches to outer side of wound spoken of above, is a small round wound discharging a similar fluid. On the left forearm over the pronator muscles, on the left shoulder-cap over the deltoid, and on the dorsum of the right forearm above the wrist, are fluctuant tumors, each the size of an egg. Plaster swathes were applied to the abdominal wounds and bandages to the tumors on the arms.

During the following three days the patient remained dull, and failed in strength. The pulse became weak and more rapid, and death occurred on the fourth day after entrance.

The temperature of the patient on the day of admission was 102° F. On the next day it was 104°, falling to normal by evening. The following morning it was 96.2°, and remained subnormal until death. The pulse was 100 on entrance, and rose gradually to 120. The respirations fell from 42 to 30.

At the autopsy no connection was found between the sinuses in the epigastrium and the thoracic or peritoneal cavity. The organs only of the case were seen by the pathologist to the hospital. The following is his description of them:

"Both lungs firmly adherent to costal pleura by old adhesions. Left lung denser than normal; does not contract on removal from thorax; the density most marked in the upper lobe. In the apex a circumscribed cavity with smooth walls filled with caseo-purulent contents; elsewhere in upper lobe

there is slaty-gray consolidation with caseation. There are also small foci of a reddish color, consolidated, from which pus can be squeezed. In the lower lobe there is much cicatricial tissue and areas of broncho-pneumonia. In the right lung there is perfect cicatrization of lower lobe with slight dilatation of bronchi. From this an abundance of pus can be squeezed, leaving very smooth cavities. In the upper lobe considerable oedema and numerous reddish foci of purulent infiltration.

"In both of the lungs there is a great deal of cicatricial tissue. Large vessels and bronchi at base free.

"Liver of ordinary size and strongly adherent to diaphragm. In the left lobe of the liver the capsule is greatly thickened, cicatricial, and apparently the entire lobe is converted into a mass of necrotic tissue infiltrated with pus. The tissue, on section, of a whitish color (no trace of liver substance apparent), and intimately united with the thickened cicatricial capsule. The right lobe of dark-red color, congested, free from abscess.

"Kidneys large, cortex hyperemic, markings normal. In both there are several circumscribed abscesses from size of a pea to a cherry. The abscesses contain pus of rather firm consistency, and a small amount of necrotic tissue. The pus is apparently infiltrated in the necrotic tissue. The pus from all these abscesses is thick and yellowish.

"In the right side of the brain, in the frontal region, at a point about corresponding to the middle of the first frontal convolution and extending down into the region of the second, there is a sharply circumscribed abscess filled with thick, greenish pus.

"Anatomical diagnosis. Tuberculosis, broncho-pneumonia, and secondary abscesses of lungs, with interstitial pneumonia. Perihepatitis and abscess of liver. Abscesses of kidneys, abscess of brain."

At both hospitals the case was regarded clinically as tubercular.

Cultures showed many colon bacilli in the heart's blood and in the abscesses in the lungs and liver. Many colonies of the staphylococcus pyogenes aureus were present in the cultures from the abscesses of the brain, lungs, and over the

deltoid. A few were found in the blood and in the abscess of the liver. A culture from an abscess of the kidney remained sterile. Coverslip preparations from some of the abscesses were reported to contain cocci and delicate bacilli.

Fortunately the abscesses of the brain and kidneys with the surrounding tissue, and considerable portions of the lungs and liver, were hardened in alcohol for microscopic purposes. Examination later showed the pathological processes to be due largely or entirely to actinomyces.

The first case of actinomycosis in man was observed by Langenbeck in 1845; but it was not until 1878 that the case, together with the original drawings, was published by J. Israel. There is also no doubt Lebert saw the organism in man in 1857, and pictured it accurately. It was not, however, until 1878 that J. Israel described a case of actinomycosis thoroughly, and showed the relation of the peculiar organism to the pathological lesions.

Actinomyces were discovered in the tongue of a cow by Hahn in 1870, and in 1877 Ballinger showed them to be present constantly in certain tumor-masses about the jaws and in the tongues of cattle. The organism was studied botanically by Harz and termed "actinomycosis bovis," or ray fungus. The identity of actinomyces in man and cattle was demonstrated by Ponfick in 1882. Since then the occurrence of the disease in man has been established by numerous observations all over the world.

In the lesions produced the actinomyces grow in characteristic colonies, which are often large enough to be recognized by the naked eye. They measure 0.18 mm. to 0.6 mm. in diameter. Occasionally they are conglomerate, and may measure as much as 2 mm., or even more, in diameter. To the naked eye the single colonies appear spherical, oval, or slightly curved; the larger colonies are more irregular. According to Bostrœm, whose monograph on this subject has become a classic, the largest colonies are found where the tissue shows the greatest inflammatory reaction.

When the growth of the colonies is not interfered with they develop, according to him, a certain typical structure which is shaped like a hollow ball with an opening at some

point, out of which streams into the surrounding tissue a network of fibres which he calls the root. The mantle of the hollow ball consists of a basement layer composed of a very thick tangle of filaments which branch dichotomously, and of collections of small round bodies. The centre of the ball consists of a few branching irregularly-arranged threads. From the basement layer arise, first, small, bush-like masses of threads but little branched; later, however, they are larger and the threads show numerous branches. On the outside lies the layer of the clubs.

The colonies are distinguished by color which depends on their age. The younger colonies appear gray and gelatinous like bits of mucus, and consist of only a loose tangle of branching filaments. In the next stage they are denser, but still small, and look grayish-white or opaque white. The denser the network of threads, the whiter and more opaque the colonies become, until finally the color passes into yellow or even brown. The clubs have a metallic lustre which may become yellowish-green or bright green if they are large and numerous.

The examination of cover-slip preparations from actively growing colonies shows often a great variety of forms, so that one is inclined to suspect a mixed infection due to several organisms. Side by side will be found coccus-like bodies, short and long bacilli, and single and branched threads, of which some stain uniformly, while others consist of longer or shorter joints each of which corresponds to a coccus or to a bacillus. Besides these there are the transparent club-shaped bodies in which can be found bacilli and cocci similar to those in the threads.

All these different forms are now pretty generally looked upon as belonging to one and the same organism, which is classed among the polymorphous bacteria. On account of the branching, Bostrœm places it in the class Cladothrix.

The club-shaped formations have always excited the greatest interest, and form one of the most characteristic features of this bacterium. According to Bostrœm, whose views are now generally accepted, these clubs are due to a peculiar degeneration of the membrane or sheath of the organism. This degeneration may occur along the course of the fila-

ments, but is found especially at the ends. The filaments can often be traced directly into them. The swollen, degenerated sheaths get denser with age; they often crack and form curious, irregular, and branching figures.

Cultures of actinomyces have been obtained by Bostrœm and by a number of other investigators. Inasmuch, however, as only those obtained by M. Wolff and J. Israel produced typical lesions when introduced into animals, it seems best to consider only their results in that direction. They succeeded in growing pure cultures from two separate cases in man, from a retro-maxillary tumor and from the lung. They found that the most important characteristics of the growth on agar were that the organism was anerobic (although not strictly so), that it showed a marked inclination to form small knots which rarely appeared before the third to the fifth day, and that these knots had a tendency to remain separate even after months. The surface growth was remarkably dry, non-elevated, and sent roots into the culture medium beneath. The older colonies were not infrequently of a pale yellow color. Microscopically the commonest form of the growth on agar was found to be a short, straight, or curved rod of which the length and breadth varied considerably. In some cultures very short thick rods were present; in others they were long, and thin or thick, with often one end swollen. Occasionally threads were found which were usually wavy or spiral.

In the cultures in eggs, although rods were present, a tangle of threads was the commonest form. The filaments were straight, wavy, or spiral, and often dichotomously divided. The filaments on the periphery of the tangled masses showed a more or less radiating pattern. The single threads were not of the same thickness: some were slender, others thick. Many of the threads could be differentiated into rows of longer or shorter rods, or into round bodies like cocci. Wolff and Israel do not agree with Bostrœm in regarding the small round bodies which look like cocci as spores, for the reason that they stain readily by Gram, a property which other spores do not possess, and they suggest that these apparent cocci may be due to division of the short rods.

No club-shaped formations could be found in the cultures.

The swollen ends of the rods often seen stained intensely with Gram, and could not therefore be due to degeneration of the sheath, for the degenerated bodies formed in this way stain only with diffuse stains.

Wolff and Israel introduced portions of their cultures into the peritoneal cavities of guinea-pigs and rabbits, and produced small tumors from the size of a millet-seed to a plum, in which were found typical colonies of actinomyces with club-shaped bodies around the periphery. The larger tumors were nodular, of a pink color speckled with yellow, and showed on section a connective-tissue capsule from which extended many anastomosing septa between the soft dough-like masses in which the colonies were situated.

There remains still to speak of the pathological lesion produced by actinomyces. This could best be studied by introducing pure cultures of the organism into animals and killing them after different periods of time. Then the direct effect of the polymorphous bacterium on the surrounding tissues could be accurately determined. This work, however, still remains to be done.

In cattle, according to Thoma, actinomyces, as a rule, give rise to circumscribed tumors in which the colonies are surrounded usually by a circular area of young, newly-formed connective tissue very rich in lymphoid and epithelioid cells. These areas, rich in cells, with the organism in the centre, are surrounded by more or less strong, at times very extensive, bands of dense fibrillar scar tissue. These tumors may resemble sarcomas. In other cases the newly-formed tissue undergoes a mucous, pus-like softening. There arise then more or less richly branched canals or fistulæ filled with mucus and purulent fluids which may permeate the organs extensively. The walls of these fistulous canals are formed by the degenerating masses of the very cellular, newly-formed tissue. This latter form of the disease is much more common in man than is the formation of tumors.

The microscopic examination of the case cited at the beginning of this paper proved extremely interesting. The denser portions of the lungs and the whole of the abscess-like mass in the liver consisted of fibrous tissue riddled with pockets of various sizes, filled with pus in which were many

colonies of actinomyces. The more normal portions of the lungs showed numerous areas of broncho-pneumonia caused by the actinomyces, colonies of which were often found in the bronchi and bronchioles. The epithelium was usually stripped from the walls, and the bronchus and adjoining alveoli were filled with polynuclear leucocytes. Often only the masses of cartilage could be found to mark the site of a bronchus.

In the abscesses in the brain and kidneys the disease showed a much more acute process than elsewhere. There was little formation of connective tissue beyond the young connective-tissue cells in the walls of the softened areas.

Through the kindness of Dr. A. K. Stone I was able to compare the character of the lesions produced in this case and the appearance of the colonies with those in a case of bovine actinomycosis. In the latter the softened areas were rather small and the production of young connective tissue abundant. The colonies were large, irregular, and showed a great many clubs all around the periphery. Even in the very small colonies the swollen sheaths around the ends of the filaments could often be demonstrated. In the human actinomycosis the areas of softening were much larger, the zone of young connective tissue thin or even in places lacking, and large colonies rather the exception. On the other hand, the production of clubs was much less marked, while the filaments often radiated out in every direction from the colonies. These filaments were found everywhere between the leucocytes, and were characterized by their wavy, spiral course, their frequently jointed appearance and their branching. Small actively-growing colonies were very abundant.

It would seem as though, in the study of the peculiar and characteristic colonies formed by actinomyces in tissues, the presence of the organism in any other form had been for the most part lost sight of. Bostrœm speaks of finding rod-shaped forms in pus cells, and suggests that they may in that way be carried to the neighboring tissues and thus infect them. Thoma calls attention to the wavy, spiral threads extending out from the colonies, and thinks that they may cause infection of the surrounding parts.

Careful study of the walls of the softened areas present

both in the human and in the bovine actinomycosis threw much light on this subject. In many of the young connective-tissue cells of the bovine actinomycosis rod-shaped forms were found singly and in clumps, lying in the protoplasm outside of the nucleus. They were frequently beaded and showed much resemblance to the bacilli of tuberculosis and leprosy in cells. Some of them seemed to degenerate and die off. Others developed into small colonies with branching filaments and gradually extended to the adjoining cells. The nuclei seemed to withdraw as far as possible from the approaching organism. Finally, the cells underwent necrosis and the nuclei disappeared. As the colonies enlarged the necrosis extended and polynuclear leucocytes began to make their appearance.

The rods were also found occasionally in the denser fibrous tissue outside of the young connective-tissue cells. In the areas of softening the same form was often found in numbers in and between the leucocytes and other cells. Branching filaments were rare.

In the human actinomycosis the process was much more rapid. There was less young connective tissue, and it broke down quickly. The actinomyces showed much less tendency to form colonies. Instead they grew luxuriantly in loose filamentous tangles and as clumps of bacilli and possibly of cocci. In the young, connective tissue were frequently giant cells which also contained the organism as rods and threads. In the brain, rod and filamentous forms were found in and between the cells outside of the area of softening. In the softened areas, rods and wavy spiral branching filaments were found in great numbers in and between the cells, and small loosely-woven colonies were very numerous.

As a colony gets larger the centre undergoes necrosis, and in this way arise the hollow-ball formations which Bostrœm regards as the highest structural development of a colony. The fact that there is often an opening through the mantle is probably due also to degeneration of the filaments at one or more points, especially on a side near another colony where the supply of nourishment would undoubtedly be less.

For staining actinomyces no method equals Gram or its modifications. The nuclei of the cells should first be stained

with lithium or alum carmine. Then every form of the organism except the clubs stands out in sharpest contrast to the rest of the tissue. It is important that the tissues be thoroughly imbedded in celloidin so that the softened areas and the colonies will not drop out of the sections. If the Gram or Gram-Günther method be employed, the sections after being decolorized and dehydrated in 95 per cent. alcohol can be cleared in oil of bergamot. After mounting on the slide every trace of the oil should be removed by xylol before adding the xylol balsam. Personally I prefer Weigert's modification, because by staining on the slide the section can usually be kept flatter.

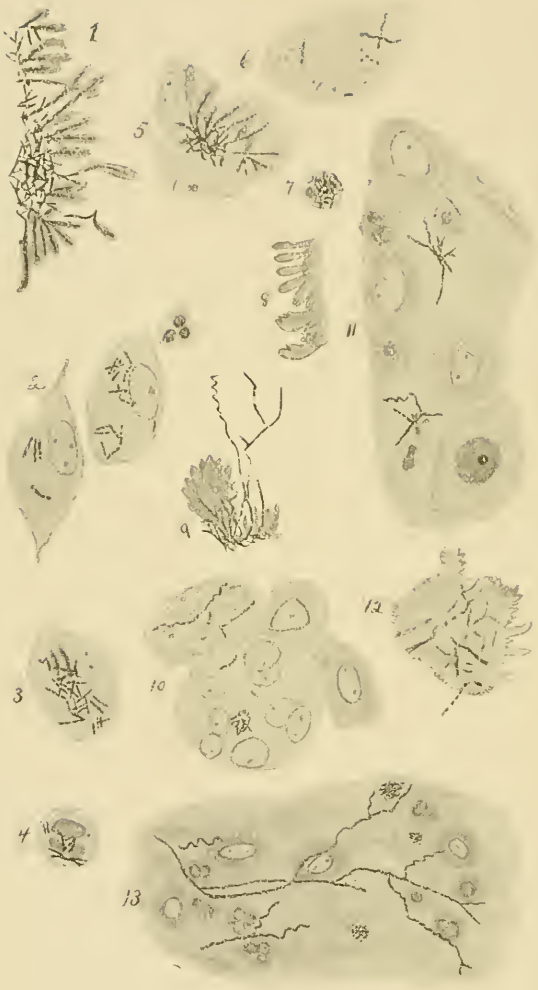
For staining the clubs in connection with the filaments in sections, the following method will be found to give quite constant results. It is Weigert's stain followed by a saturated solution of fuchsine in aniline oil:

(1) Aniline oil gentian violet, at least ten minutes.
(2) Iodine solution, 1–2–100 one to two minutes.
(3) Fuschsine (saturated solution in aniline oil), two to five minutes or more.
(4) Aniline oil. Change several times; watch the process of decolorization with low power; when sufficient, wash off the aniline oil with xylol.
(5) Xylol balsam.

The section should be dried with filter-paper between each step. The nuclei will appear sharper if the sections are first lightly stained with alum cochineal, which gives the best contrast. The blue filaments within the swollen red sheaths can be best seen in the young colonies of bovine actinomycosis.

Infection in man with actinomyces occurs in four ways: by the mouth and pharynx, by the respiratory tract, by the gastro-intestinal tract, and much more rarely through the skin. In a number of cases, both in man and in cattle, the source of the infection has been demonstrated to be some portion of an ear of grain on the surface of which the acitnomyces could be shown to be present.

The case here reported would seem to have been infected through the respiratory tract. From there the process extended to the left lobe of the liver. The other organs and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probably the arms were infected through the blood. Although not demonstrated, it is probable that the process extended from the lungs to the abdominal wall, and that in this manner the abscess in the epigastrium arose, for it is characteristic of actinomycosis of the lungs that it has a tendency to extend to the chest wall and then to appear beneath the skin.

Actinomycosis of the brain is quite a rare lesion. Martin has just published, from Chiari's laboratory, two cases which he studied there. He was able to find record of but four others.

It has been claimed that there are a great many varieties of actinomyces. Gasperini enumerates eighteen. This is a question which, together with several other disputed points in regard to actinomycosis, will have to be left to the future to decide.

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Explanation of Figures in Plate.

Figs. 1 to 5 are from a case of bovine actinomycosis.

Figs. 6 to 13, from the case reported in the paper.

Fig. 2 shows the rod forms in the young connective-tissue cells around an area of softening.

Figs. 3 and 5 show the development of a young colony in the epithelioid cells of the young connective tissue.

Fig. 4, a leucocyte containing rod forms.

Fig. 1, the edge of a young colony showing the swollen sheaths around the ends of the filaments. The same condition is seen in Figures 3 and 5.

Figs. 6 and 10, giant cells from the wall of the abscess of the brain with rods and filaments.

Fig. 11, brain tissue just outside of the wall of the abscess showing branching filaments and two degenerating rods.

Fig. 9, edge of a colony in the liver showing the filaments extending out beyond the clubs.

Fig. 7, leucocyte from lung filled with rods.

Fig. 13, branching filaments and small clumps of bacilli and perhaps of cocci in softened area of liver.

Fig. 8, small mass of clubs, some of which are fissured, left after death of colony; from liver.

Fig. 12, section through surface of old colony of liver showing a fused mass of clubs and a few degenerating rods and filaments.

The drawings were all made with a Zeiss one-twelfth oil immersion and No. 4 ocular.

XXI.

A CLINICAL AND EXPERIMENTAL STUDY OF THE LEUCOCYTOSIS OF DIPHTHERIA.*

By John Lovett Morse, M.D.

A great deal of work has been done during the last few years on the subject of leucocytosis, especially in acute diseases. Diphtheria, however, has been rather neglected. As it is an acute disease with marked constitutional symptoms due to the absorption of the toxic products of bacterial growth, a leucocytosis is to be expected. The observations hitherto reported have shown this to a marked degree.

Bouchut and Dubroisay[1] in 1877 made repeated examinations of the blood by Hayem's method, in 84 children sick with diphtheria, and obtained 4,305,000 as the mean of 93 counts of the red corpuscles; that is to say, a moderate diminution, which was the greater the severer the sickness and the higher the fever. The white cells were, in the average of 93 counts, increased to 26,824, making the ratio of white to red equal 1 to 160. Bouchut gives the following summary regarding the frequency of the occurrence of the various numbers. The white corpuscles amounted to:

0 to 5,000	in 1 case.
5,000 to 10,000	in 11 cases.
10,000 to 20,000	in 28 cases.
20,000 to 30,000	in 19 cases.
30,000 to 40,000	in 15 cases.
40,000 to 50,000	in 7 cases.
50,000 to 60,000	in 9 cases.
60,000 to 100,000	once in every 10 cases.

The very high grade of leucocytosis reported by these observers seems almost incredible, but is probably to be

* A paper to which was awarded the First Lyman Prize for the year 1894. From the pathological laboratories of the Boston City Hospital and the Harvard Medical School.

explained by the fact that they used Hayem's apparatus, which does not compare in accuracy with the Thoma-Zeiss. It is also possible that owing to the lack of bacteriological examinations, there may have been some errors in diagnosis.

In a later communication Bouchut[2] reports the results of 177 observations in severe "septicemic diphtheria." He found an "acute leucemic" condition of the blood, which increased with the severity of the disease and diminished again with an improvement. He did not find this marked leucocytosis in the lighter cases, however. Hence he considers the examination of the blood as regards the number of the white corpuscles as of importance for diagnosis. No differential count of the white corpuscles was made in these cases, of course.

The only other examination of the blood in diphtheria which I have been able to find reported in an extended search through the literature of leucocytosis is one by Reinert.[3] He studied one case in a twenty-four-year-old girl, where a blood examination was made, on account of an ulcer of the stomach, before the attack of diphtheria. His results were as follows:

11, xii, 3 P.M. Red 4,772,000.
13, xii. Patient sickened with diphtheria with moderate fever.
16, xii, 3.30 P.M. Red, 5,050,000; white, 5,763; white to red as 1 to 870.
19, xii, 3.30 P.M. Second day after cessation of fever. Red, 4,398,000.
19, xii, 4.30 P.M. Red, 4,732,000; white, 7,700; white to red as 1 to 610.

That is, a slight increase of the colored elements was observed during the fever period, "which may be easily explained by the diminished ingestion of food and drink." There was little or no increase in the leucocytes.

As the results hitherto obtained were rather surprising and lacked confirmation, there seemed to be room for further investigation. Moreover, many points had not been studied, as the character of the leucocytosis, its influence on prognosis, and its relation to the membrane, glands, and complications. Through the kindness of Drs. Buckingham and Folsom I have been enabled to examine the blood of 30 cases in the diphtheria ward of the Boston City Hospital. Owing to the

lack of time, in but one case was more than a single examination made. The presence of the Klebs-Loffler bacillus had been previously demonstrated in all of them, and none of them were complicated by other diseases, acute or chronic. The effect of the symbiosis with other bacteria was not investigated.

The blood was in every case taken from the lobe of the ear and examined with a Thoma-Zeiss apparatus. It was diluted 1 to 200 with a three-per-cent. salt solution colored with methylene-blue, and the corpuscles in one cubic millimeter counted. Cover-slips were in all cases made at the same time, hardened by heat or benzine, and stained with Ehrlich's "triple-stain." A differential count of from 300 to 500 white corpuscles was then made, the classification recommended by Ehrlich being used. As far as possible, the blood was taken at the same time of day and between three and four hours after food. The condensed results of my work are appended in the accompanying table, which is arranged according to the day of the disease.

It is evident from the above table that diphtheria is accompanied by a marked hyperleucocytosis, larger even than that found in pneumonia. This has become well marked by the third day, and is probably present earlier. In a general way it increases as the disease progresses, is greatest at the height of the disease, diminishes during convalescence, and disappears with or soon after the membrane. It also in a general way corresponds with the amount of membrane, varying with it directly. There are notable exceptions to this rule, however, as in Case VII., where the throat was full of membrane and only 8,000 white corpuscles were found, and in Case V., where there were 42,000, but only a moderate amount of membrane. There seems to be no evident connection between the glandular enlargement and the increase in white corpuscles. It is noticeable, however, that the fatal "septic" cases with greatly enlarged glands all showed a very marked hyperleucocytosis. Some of the mild cases, nevertheless, with little or no enlargement of the glands, showed just as much. The condition of the lungs and kidneys apparently had no influence in determining the number of white cells. It thus becomes evident that the hyperleucocytosis is

No.	Age.	Day of Disease.	Hours since Food.	Temperature.	Membrane.	Glands.	Lungs.	Albumin.	Condition.	Result.	Remarks.	Red.	White.	Ratio.	Small Lymphocytes.	Small Lymphocytes, Transition Forms.	Polynuclear Neutrophiles.	Eosinophiles.
1	23	3	2	N.	Slight; mostly nasal	None	Normal	None	Good	Well		5,786,000	9,000	1 to 643	15%	4	76%	5%
2	12	3	4	100°	Moderate	Small	Normal	None	Very good	Well		4,674,000	18,000	1 to 260	8	5	85	2
3	9	3	4	N.	None; nasal	Small	Normal	None	Very good	Well		4,640,000	14,000	1 to 331	10	3	76	8
4	29	3	3½	99°	Moderate	Small	Normal	None	Very good	Well	Height of disease	5,564,000	16,000	1 to 348	16	4	79	1
5	27	3	1	100.5°	Moderate	None	Normal	None	Good	Well	Height of disease	5,618,000	42,000	1 to 134	15	6	78	1
6	27	5	2	99°	Very little	None	Normal	None	Good	Well	Convalescing	5,262,000	17,000	1 to 303	17	6	76	1
7	13	5	4½	100.4°	Much	Large	Normal	⅛%	Good	Well		5,536,000	8,000	1 to 690	25	5	69	1
8	4½	5	3½	N.	Moderate	Moderate	Normal	Slight trace	Fair	Well		5,200,000	20,000	1 to 260	11	6	80	3
9	8	5	Eating	99°	Moderate	Small	Normal	None	Good	Well		5,044,000	28,000	1 to 180	11	4	83	2
10	15	5	4	100.4°	Much	Large	Bronchitis	⅛%	Fair	Died in 4 days	Became septic	5,752,000	28,000	1 to 205	11	4	35	6
11	30	6	4	100.4°	Little	Slight	Normal	None	Good	Well	Convales'g. Anemic	3,312,000	20,000	1 to 165	9	4	84	3
12	3	6	Not Eating	99°	Very much	Very large	Broncho-pneumonia	Trace	Moribund	Died in 5 hrs.	Septic. Throat and nose "rotten"	5,728,000	40,000	1 to 143	10	6	82	2
13	12	6	4½	99.8°	Little	Small	Normal	None	Good	Well	Cardiac souffle	5,196,000	24,000	1 to 216	17	4	75	4
14	3½	6	½	N.	Much	Considerable	Slight bronchitis	None	Fair	Well	Obstruction	5,224,000	48,000	1 to 109	4	2	94	0
15	5½	6	Vomiting	99°	Much	Very large	Normal	None	Moribund	Died in 4 hrs.	Septic. Hemorrhagic	5,300,000	20,000	1 to 265	6	2	60	0
16	4	6	2½	N.	Much	Small	Normal	None	Fair	Well	Slight obstruction	5,164,000	32,000	1 to 161	10	9	81	0
17	9	6	1½	101.2°	Much	Very large	Bronchitis	Slight trace	Bad	Died in 22 hrs	Septic	4,910,000	42,000	1 to 117	8	3	88	1
18	52	7	4	102.4°	Much	Small	Bronchitis	None	Good	Well		4,656,000	20,000	1 to 233	8	4	88	0
19	2	7	4	97°	Slight	Small	Slight bronchitis	None	Good	Well		5,704,000	12,000	1 to 475	30	7	59	4
20	8	7	3	N.	Moderate	Moderate	Normal	Slight trace	Good	Well	Convalescing	5,412,000	20,000	1 to 270	9	7	80	4
21	27	8	4½	N.	Much	Slight	Normal	⅛ to ¼%	Fair	Well		6,860,000	40,000	1 to 171	9	3	84	4
22	22	8	2½	N.	Much	Small	Normal	⅛ to ¼%	Good	Well		6,068,000	20,000	1 to 303	12	4	83	1
23	9	8	½	99°	Moderate	Small	Normal	None	Good	Well		4,484,000	24,000	1 to 187	16	5	74	5
24	30	9	3	99.4°	Very little	None	Normal	None	Good	Well		5,138,000	12,000	1 to 428	16	7	75	2
25	7	10	4½	N.	None for 2 dys.	Very small	Normal	None	Good	Well	Convales'g. Still K.I.	4,158,000	8,000	1 to 513	19	4	75	2
26	7½	10	3	100°	Little	Moderate	Normal	None	Good	Well	Convalescing	4,770,000	10,000	1 to 477	16	6	77	1
27	24	11	4	N.	Slight	Slight	Normal	None	Good	Well	Convalescing	5,012,000	12,000	1 to 417	23	6	68	3
28	25	13	3	N.	Little	Small	Normal	None	Good	Well	Convalescing	5,625,005	15,000	1 to 375	30	8	59	3
29	24	14	4	N.	Little	None	Normal	None	Good	Well	Convalescing. Memb kept up by H_2O_2	5,240,000	16,000	1 to 327	10	3	85	2
30	23	21	4	N.	None for 2 dys.	Small	Normal	None	Good	Well	Convales'g Still K.I.	4,608,000	8,000	1 to 576	32	6	58	4

not due to any symptom or combination of symptoms, but is the result of some general influence which is present in every case. This influence can be no other than that of the toxines absorbed. Whether the individual variation is due to the amount of absorption, to some difference in the virulence of the toxine absorbed, or to some difference in the resistant powers of the person attacked, must, in the present state of our knowledge, be left unanswered. In the great majority of cases the increase was in the polynuclear neutrophiles, that is, the typical leucocytosis. In several, however, the proportions closely approach the normal. In a few, chiefly convalescent cases, there seemed to be a lymphocytosis; this, too, in cases without much external glandular enlargement.

The number of the erythrocytes was, with one or two exceptions, always somewhat above normal. The only patient in which they were diminished was a woman, evidently subject to a chronic anemia. I am unable to give any explanation of this increase.

This study establishes the fact pretty clearly, I think, that the examination of the blood in diphtheria as to leucocytosis is of no value in prognosis. For, although fatal cases have a pretty marked one, yet it is almost always present, and often very pronounced, even in the mildest cases.

Having thus established the existence of a leucocytosis in diphtheria, I will now take up briefly the various views with regard to the origin of leucocytosis in general, and later detail the results of some experiments in animals which I have made in this connection. I will take up the theory which seems to me most probable last, that of Goldscheider, comparing his views with the others in turn and quoting largely from him.

Von Limbeck[4] considers that leucocytosis stands in the closest connection with the formation of exudate, and considers it as entirely secondary to the exudation. He found that when the toxic products of micro-organisms were introduced into animals, a reaction followed which corresponded to the size of the dose; that neither blood nor other tissues reacted to small amounts; and that after larger, a local reaction and proportionate hyperleucocytosis occurred. A much more significant reaction of the tissues and a greater

hyperleucocytosis followed when living micrococci were introduced. He concludes that the hyperleucocytosis arises from the development or presence of bacterial products in the organism, which have a further action on the white cells and bring them into the circulation. He does not explain what this action is, and expresses no opinion as to whether those cells are new-formed or merely driven out of the blood-forming organs. He nevertheless calls attention to chemotaxic action as probably playing a rôle in the process.

His theory must be considered as of little value, however, as he did not recognize the hypoleucocytosis which has been found by other observers. His failure to find this was undoubtedly because he made no examination under forty-eight hours. Moreover, other investigators, notably Goldscheider and Jacob, have produced a marked hyperleucocytosis by the subcutaneous injection of organic extracts without producing any swelling or exudate. These experiments evidently destroy the foundations of von Limbeck's theory.

Römer,[6] as the result of his own and Buchner's[7] work, concludes that the destructive products of dead bacteria or other cell-life, when introduced into the circulation, exert a direct formative action on the white cells, causing a new formation of leucocytes, exclusively in the venous blood. This increase arises through amitosis. He also claims that the cells found increased may in part be already present in the blood-making organs and be brought into the circulation by chemotaxic action. Rieder[8] and Löwit completely contradict his experiments and arguments, while Goldscheider and Jacob find no difference in the blood of the veins and arteries, and show conclusively that after a short time there cannot be. Their experiments show that there is no such thing as hyperleucocytosis in the veins alone, which could only occur as the result of a direct new formation of white cells in the circulating blood. Both Löwit and Goldscheider and Jacob deny that this takes place. They both find immediately after the hypoleucocytosis almost exclusively mononuclear cells. Gradually, however, during the course of the hyperleucocytosis, a decided preponderance of polynuclear arises. Goldscheider and Jacob also lay much stress on the fact that most of these cells are eosinophile, and hence consider that

they are not entirely formed from the new mononuclear, but are partly old cells which were lying in the bone-marrow ready for extrusion and have been brought into the circulation in increased numbers as the result of the chemical irritation of the injected material.

Löwit considers hyperleucocytosis to be entirely independent of chemotaxis, and believes that it is due solely to a previous impoverishment of the blood in leucocytes. He also believes that a hyperleucocytosis occurs every time after a part of the leucocytes have perished, because an increased influx of young leucocytes out of the blood-forming organs is thereby brought on, that is, hyperleucocytosis follows leucolysis, which may result from the presence of certain substances, as bacteriological products, in the blood. He found that in all cases immediately after the injection there was a decided diminution in the leucocytes, but that then a gradual increase took place, which developed as much more intensely as the previous diminution was great. Thus, according to Löwit, hypoleucocytosis and hyperleucocytosis are two processes which are inseparably connected, the second occurring as the result of the first and being impossible without it. Goldscheider and Jacob were able, however, by the use of repeated small injections, to produce a primary hyperleucocytosis. They also showed, by autopsies on animals killed during the stage of hypoleucocytosis that the leucocytes were accumulated in the capillaries of the lungs and not destroyed. Löwit denies chemotaxis any part in the production of leucocytosis, but recognizes, however, that he cannot explain how leucolysis causes such a marked discharge of young cells from the blood-forming organs. It is difficult to see on what grounds he can deny the influence of chemotaxic action upon this process.

Schultz [10] believes that in all conditions which are considered as hypoleucocytosis and hyperleucocytosis the white cells are neither diminished nor increased, but are merely distributed differently in the vessels. According to his experiments with protein and other injections, there is neither a destruction of leucocytes — Löwit's leucolysis — nor an absolute increase of the leucocytes from the blood-forming organs afterward. That is, he believes that the hypoleu-

cocytosis is merely due to the accumulation of the white cells in the central vessels with a corresponding diminution of those in the peripheral, the hyperleucocytosis being due to the reversal of these conditions, the cells simply being differently distributed. Goldscheider and Jacob, however, found that the cells which accumulated in the capillaries of the lungs remained there, even increasing in number during the early stage of the hyperleucocytosis, thus showing that there is no shifting of the cells, but an absolute increase. This increase is sufficient to counterbalance the first loss of cells from the general circulation and still far exceed the number normally there.

Goldscheider and Jacob[5] have performed a very large number of very careful injection experiments on rabbits, using extracts of various organs and bacterial products. Many of their results have been already noted. They made counts immediately after injection and then at frequent intervals. After injections into veins they found in one case a marked hypoleucocytosis in twenty-five seconds. On the average the hypoleucocytosis reached its minimum in five or ten minutes; then the number began to increase, the maximum hyperleucocytosis usually being reached in five hours, but often only after twelve to eighteen hours; the normal was again reached in twenty-four to forty-eight hours. After subcutaneous injections the results were the same, but slower in making their appearance, the minimum appearing in three or four hours, the earliest maximum in fourteen to eighteen hours. Autopsies on animals killed during hypoleucocytosis showed the capillaries of the lungs to be full of leucocytes; there were as many, or more, in them during the hyperleucocytosis. They also succeeded, by giving repeated small injections, in producing a primary hyperleucocytosis. In other cases, by using very large doses, they caused death while the hypoleucocytosis still persisted.

Müller,[11] Everard, DeMoor, and Massart,[12] and Canthack[13] have obtained similar results as regards hypoleucocytosis and hyperleucocytosis. Everard sums up his work as follows: The injection of bacterial cultures, living or dead, results first in a diminution of the circulating leucocytes, especially of those with multiple nuclei and granular pro-

toplasm. When the animal survives the infection, the stage of hypoleucocytosis is followed by one during which the polynuclear granular cells are very abundant; then the blood resumes its normal characteristics. The hyperleucocytosis is not typical in cases which succumb to the injection, or may be entirely wanting in those which die quickly. Sometimes, when the infection is prolonged, it is replaced by a series of oscillations.

Canthack made hourly examination of the blood after the injection of filtered cultures of vibrio Metchnikovi. He found, at first, during the rapid rise of temperature, a marked diminution of the leucocytes which persisted until the pyrexia passed its climax. Four to six hours after, the number was enormously increased, reached its height at the ninth hour, but persisted, though to a less degree, for forty-eight or seventy-two hours, or even longer.

As the result of their experiments, Goldscheider and Jacob conclude that the phenomena of leucocytosis are due to chemotaxic action. The first action is a repellent one on the leucocytes, and they are arrested and held in the lung capillaries, where they remain. The second is irritative — attractive — on the blood-forming organs, and the white cells already formed there are set free and an increased number of new ones formed and also set free. Some of these also are arrested in the lungs as the result of continued repellent action. There is no instant leucolysis, and the process is something more than a mere changing of leucocytes, without increase, from the inner vessels to the outer, and *vice versa*. They explain the hyperleucocytosis without hypoleucocytosis which occurs after small doses by the fact that the small quantity, being distributed throughout the whole circulating fluid, is not sufficiently concentrated to exert an energetic enough repellent action on the leucocytes to cause much retention in the capillaries. If, however, such small doses are repeated at certain intervals, the positive, or attractive, influence gradually increases, finally becoming far superior to the negative influence which is exerted by these small doses. Thus the equilibrium between the two processes is finally destroyed in favor of the first, the result being a decided hyperleucocytosis. They

explain death during the hypoleucocytosis by the continuation of the repellent action, the irritative action being so strong as to paralyze the blood-forming organs.

Several difficulties confront us when we attempt to compare the leucocytosis in man with that observed in animals. We naturally cannot subject a sick man to so frequent blood-examinations as an animal; and as the disease often extends over days and weeks, important changes may easily escape observation. Moreover, we almost never get a blood-examination in the early stage of a disease. However, the vast number of blood-examinations which have been made clinically during the past few years have given us a general idea of the leucocytosis in most diseases. A hyperleucocytosis has been demonstrated in most acute infectious diseases, especially febrile.

A constant diminution has been found only in typhoid, malaria, and puerperal sepsis. There is said to be a decided hypoleucocytosis in the acute infectious diseases where the patients die within a few days. This is certainly the case in pneumonia. I have not observed it in diphtheria, the only case which had a normal number of white cells recovering.

It is possible that there may be a primary hypoleucocytosis in man, but, as already noted, the patient is almost never seen early enough to demonstrate this, if it exists.

It has been shown above, however, that it is possible experimentally to produce a primary hyperleucocytosis. Now the conditions in man are essentially the same as those in the experiments made regarding this point. For the ordinary method of infection is not by the sudden entrance of a large amount of toxic substances into the circulation, but by the continuous absorption of small quantities. Hence we expect a hyperleucocytosis without a previous hypoleucocytosis. If the infection be extremely sudden and severe and rapidly fatal, we have a condition analogous to that in animals killed by a very large dose during hypoleucocytosis. That is to say, the toxic material is sufficiently concentrated to paralyze the blood-forming organs. This would explain the fatal cases of pneumonia with no increase of white cells. Thus it is evident that the chemotaxic theory explains satisfactorily the leucocytosis found in man as well as in animals.

A series of experiments was made on animals to determine primarily whether the hyperleucocytosis observed clinically in diphtheria was due to the Klebs-Löffler bacillus and its toxines, or to associated bacteria. As far as I know, these are the first injection experiments ever made on animals with this bacillus or its products. At the same time a careful study of the leucocytosis was made, and the work of previous investigators in this line gone over. Autopsies were made in all cases and the organs examined.

Rabbits were used, as their ears naturally fit them for intravenous injections and the blood is easily obtained. The normal number of white corpuscles averages a little higher than in man. As there is a considerable variation in individual animals, a count was made in every case before beginning an experiment. Rabbits offer the further advantage that they present no digestion leucocytosis.[10] The blood was always taken from the ear and diluted (1 to 200) with a three-per-cent. salt solution colored with methylene blue. The Thoma-Zeiss instrument was used, and the corpuscles in at least fifteen cubic millimetres counted. The differential counts were of 500 white cells hardened with alcohol and ether and stained with Ehrlich's "triple-stain." The toxines used were from a filtered bouillon culture which had been twenty-three days in the thermostat, and was preserved by the addition of one-half per cent. of carbolic acid.

I wish here to acknowledge my indebtedness to Dr. J. H. Wright, of the Harvard Medical School, for the use of the toxine solution, and to thank both him and Drs. Councilman and Mallory for valuable advice and assistance in my work.

The object of the first experiment was to determine whether a local diphtheria would cause a hyperleucocytosis.

Rabbit No. 1.

October 24. 9.30 A.M., white corpuscles, 5,800. 11 A.M., the palpebral conjunctiva of the left eye was excoriated over a small area, and a pure, fresh culture of the Klebs-Löffler bacillus on blood-serum rubbed in. This culture was the second generation from a fatal case.

October 25. 9.30 A.M., a spot of membrane, about one-quarter of an inch long and one-sixteenth of an inch wide, was present at the site of the excoriation. White corpuscles, 6,400.

October 26. 10.30 A.M., membrane all gone. White corpuscles, 7,100. As the amount of membrane had been so small, and the increase

of white corpuscles so slight as to be within the bounds of possible error, it seemed best to repeat the experiment. Accordingly, the conjunctiva was again excoriated at the same spot, but more deeply and over a larger area. 11.30 A.M., reinoculated with same culture.

October 27. 10.15 A.M., excoriated spot covered with membrane over an area about one-quarter of an inch square, the membrane also extending a little on to the ocular conjunctiva which had not been irritated. White corpuscles, 9,100.

October 28. 9.15 A.M., membrane a little less extensive. White corpuscles, 9,100. A blood-serum tube, inoculated from the eye at this time, twenty-four hours later showed a pure culture of the Klebs-Löffler bacillus.

October 29. 12 M., membrane only about half as extensive. White corpuscles, 8,400.

October 30. 9.30 A.M., membrane disappeared. White corpuscles, 8,200.

November 1. 9.30 A.M., white corpuscles, 6,000.

The result of this experiment proves that a local diphtheria, produced by the Klebs-Löffler bacillus alone, is capable of causing a hyperleucocytosis. The results also agree in a general way with the conclusions arrived at clinically; namely, that the hyperleucocytosis varies with the amount of membrane and ceases soon after its disappearance.

A series of experiments was next undertaken to determine if the toxine produced by the Klebs-Löffler bacillus would alone, as seemed probable, cause a hyperleucocytosis.

Rabbit No. 2.

October 31. 9.30 P.M., white corpuscles, 12,400.

November 1. 10 A.M., one-half a cubic centimetre of toxine solution injected into ear-vein.

November 2. 9 A.M., found dead. A count of the heart's blood showed white corpuscles, 23,400. Autopsy showed inguinal, axillary, mesenteric, and retroperitoneal glands enlarged and reddened. Liver, macroscopically, showed no necrosis. Kidneys moderately congested. No fluid in thorax. Lungs somewhat congested and slightly edematous. Bone-marrow somewhat reddened.

Rabbit No. 3.

November 2. 12 M., white corpuscles, 6,700. 12.45 P.M., one-tenth of a cubic centimetre of toxine solution injected into ear-vein.

November 3. 10.30 A.M., white corpuscles, 12,000.

November 4. 10 A.M., white corpuscles, 16,300. At this time the animal was decidedly sick, showing marked paresis of hind legs and some of front. 3.30 P.M., white corpuscles, 20,800. The rabbit was moribund at the time of this examination.

November 5. 12 M., autopsy, rabbit having died during the night, showed axillary glands enlarged and reddened. Little or no enlargement and no injection of inguinal, mesenteric, or retroperitoneal glands. Suprarenals much reddened. No macroscopic necrosis of liver. Kidneys much congested. No fluid in thorax. Lungs slightly congested, but not edematous. Bone-marrow very red.

In this case the leucocytosis had doubled in twenty-two hours and continued to increase steadily up to the time of death.

RABBIT NO. 4.

November 6. 3.30 P.M., white corpuscles, 5,200. 4.30 P.M., one-fiftieth of a cubic centimetre of toxine solution injected into ear-vein.

November 7. 9.30 A.M., white corpuscles, 8,400. 3.30 P.M., white corpuscles, 11,400. The rabbit was now evidently sick.

November 8. 9.15 A.M., white corpuscles, 11,700. Rabbit much sicker. A differential count of the white cells made at this time, the height of the hyperleucocytosis, resulted as follows:

	Per cent.
Small Lymphocytes	30.8
Large Mononuclear and Transition Forms	5.8
Polynuclear Neutrophiles	7.8
Eosinophiles	55.6

The consideration of this, as well as of the following differential counts, will be taken up later.

November 9. 9.30 A.M., white corpuscles, 7,600. The rabbit was livelier than during the two previous days, and evidently on the road to recovery.

DIFFERENTIAL COUNT.

	Per cent.
Small Lymphocytes	41
Large Mononuclear and Transition Forms	10
Polynuclear Neutrophiles	11
Eosinophiles	38

November 10. 9.30 A.M., white corpuscles, 4,000. Rabbit still improving.

November 17. 9.30 A.M., white corpuscles, 5,800. The rabbit was now entirely well, but showed considerable induration about the seat of injection. It showed no signs of paralysis at any time.

In this case the increase in white corpuscles was well marked in seventeen hours, reached its maximum in forty-one hours, and had disappeared in eighty-nine hours. It is to be noted that the animal's condition varied with the

leucocytosis, he being sickest when it was at its height, and improved as it diminished. It is also worth noting that the hyperleucocytosis disappeared before any local reaction manifested itself, which is in direct contradiction to v. Limbeck's theory.

The three preceding experiments seem amply sufficient to prove that the toxines alone are competent to produce a hyperleucocytosis. They also justify the conclusion that the hyperleucocytosis in diphtheria is due to the absorption of the toxic products of the Klebs-Löffler bacillus and varies directly with the amount of absorption.

It is probable, however, that clinically the toxic products of associated bacteria play a part in its production. The subsequent experiments, although undertaken for other purposes, emphasize the same points.

The next experiment was undertaken to determine whether the toxines of the Klebs-Löffler bacillus cause an initial hypoleucocytosis, and if so, the time of its appearance and its duration.

RABBIT NO. 5.

November 10. 4 P.M., white corpuscles, 7,900.

DIFFERENTIAL COUNT, NORMAL BLOOD.

	Per cent.
Small Lymphocytes	45.2
Large Mononuclear and Transition Forms	6.6
Polynuclear Neutrophiles	11.2
Eosinophiles	37.0

November 11. 9.15 A.M., four-tenths of a cubic centimetre of toxine solution injected into ear-vein. 9.30 A.M., white corpuscles, 5,100.

DIFFERENTIAL COUNT.

	Per cent.
Small Lymphocytes	32.2
Large Mononuclear and Transition Forms	7.6
Polynuclear Neutrophiles	8.9
Eosinophiles	51.3

10.30 A.M., white corpuscles, 4,900.

DIFFERENTIAL COUNT.

	Per cent.
Small Lymphocytes	31.2
Large Mononuclear and Transition Forms	7.4
Polynuclear Neutrophiles	11.2
Eosinophiles	50.2

11.45 A.M., white corpuscles, 7,300. 3.30 P.M., white corpuscles, 13,100.

November 12. 9.15 A.M., white corpuscles, 11,500. The animal at this time, although not well, showed no very marked symptoms.

November 13. 9 A.M., found dead, but warm.

At the autopsy the axillary glands were slightly enlarged, but not much reddened. No enlargement or injection of inguinal, mesenteric, or retroperitoneal glands. Suprarenal capsules moderately reddened. No macroscopic or microscopic necrosis of liver. Kidneys much congested. No fluid in thorax. Lungs moderately congested, but not edematous. Bone-marrow very red.

As is evident above, the result of this experiment is positive, a definite hypoleucocytosis being present fifteen minutes after the injection. The lowest point was reached in an hour and a quarter. One and one-quarter hours later the number had nearly returned to normal, and three hours after this there was a marked hyperleucocytosis. The differential counts will be considered later. In "Rabbit 7," also, a marked hypoleucocytosis was present in half an hour.

The existence of hyperleucocytosis and hypoleucocytosis having been demonstrated, a series of investigations was begun to determine, if possible, the nature of the processes, and to determine, also, which, if any, of the theories detailed above was tenable. The best way of getting at this seemed to be to kill animals during the various stages, and then to make careful microscopic examinations of the organs, paying especial attention to the distribution of the white cells in them and to the bone-marrow.

The animals were killed by a sharp blow on the back of the neck, immediately autopsied, and the organs removed with as little handling as possible and placed in a saturated solution of corrosive sublimate in normal salt solution. After hardening in this for twenty-four hours, they were washed in running water for twenty-four hours, then placed in absolute alcohol and mounted in celloidin in the usual manner. The sections were all cut of the same thickness in order to avoid error. Sections of the femur were made in the normal rabbit and in one killed during hyperleucocytosis, and kept in absolute alcohol for ten days. The outside bone was then removed, and the marrow placed in five-per-cent. nitric acid for twenty-four hours. It was then washed and

mounted in celloidin as were the other organs. Cover-slips were also made from the fresh bone-marrow and hardened in alcohol and ether.

RABBIT NO. 6.

A normal rabbit killed for purposes of comparison. It is noteworthy that the bone-marrow of the rabbit is extraordinarily rich in eosinophile cells, they forming the very great majority of the white cells present there.[14]

RABBIT NO. 7.

Killed during hypoleucocytosis.

November 15. 10.30 A.M., white corpuscles, 7,300. 11.15 A.M., seven-tenths of a cubic centimetre of toxine solution injected into ear-vein. 11.45 A.M., white corpuscles, 4,500. 12.15 P.M., killed.

At the autopsy no microscopic changes were recognizable. Bone-marrow, yellowish-red. Microscopical examination of specimens of the liver and kidney, stained with saffranin, showed no abnormal changes. There was no congestion and no increase in the number of white cells in their capillaries. Sections of the lungs were stained both with saffranin and with Ehrlich's "triple-stain," the latter giving a most beautiful differentiation of the white cells. These sections were carefully compared with those of the normal lung prepared in the same way. It must be admitted here that the conclusions arrived at are founded entirely on individual estimation and judgment, it being impossible to count the cells.

Drs. Councilman and Mallory, however, who were shown the specimens from the three cases, without being told which was which, arrived at the same conclusions regarding the number of white corpuscles as myself. While there was no noticeable increase in the number of red corpuscles in the lung capillaries, the white cells were very much more abundant than in the normal lung. In the large blood-vessels, however, the white corpuscles were much diminished. It was impossible to tell whether any one kind of white cells was more than proportionately abundant, the general impression being, however, that the proportions were about normal. Numerous hyaline thrombi were present in the lungs.

RABBIT NO. 8.

Killed during hyperleucocytosis.

November 18. 11 A.M., white corpuscles, 12,700. 11.45 A.M., two-tenths of a cubic centimetre of toxine solution injected into ear-vein.

November 19. 9.30 A.M., white corpuscles, 29,200.

Differential Count.

	Per cent.
Small Lymphocytes	15.4
Large Mononuclear and Transition Forms	1.6
Polynuclear Neutrophiles	4.4
Eosinophiles	78.6

11 A.M., killed.

At the autopsy the axillary, inguinal, mesenteric, and retroperitoneal glands were unchanged. Suprarenals much reddened. No macroscopic necrosis of liver, which was somewhat congested. Kidneys much congested. Urine not hemorrhagic. No fluid in thorax. Lungs moderately congested, but not edematous. Bone-marrow yellowish-red, but no redder than normal.

Microscopic examination of sections of the liver, stained with saffranin, showed marked congestion. The increase in white cells did not seem out of proportion to the increase in red, when the hyperleucocytosis was taken into consideration. Double-staining with eosin and methylene-blue failed to show any necrotic areas. Sections of the kidney, stained with saffranin, showed an enormous congestion. The increase in white cells, however, was not out of proportion to the hyperemia and hyperleucocytosis. Sections of the lungs were prepared as in the previous cases. There was a decided increase in the number of red cells in the capillaries. The white corpuscles seemed less abundant in the capillaries than during hypoleucocytosis. They were more numerous than in the normal lung, however, and seemed to be in larger numbers than the congestion and general hyperleucocytosis would explain. The impression obtained was that the eosinophile cells were more than proportionately abundant. The white cells in the large blood-vessels seemed about proportionate to the red corpuscles, when the general hyperleucocytosis was taken into consideration. Numerous hyaline thrombi were present in these lungs also.

Specimens of the bone-marrow stained with saffranin and compared with the normal showed a diminution in the number of polynuclear cells and giant cells; nuclear figures, however, were much more abundant. Those stained with "triple-stain" also showed a diminution in the number of giant cells. There was a great increase, however, in the number of small cells with small, round, deeply staining nuclei and a marked increase in the nuclear figures in these same cells. I am unable to give any explanation of the significance of these cells or of the change in them.

The object of the last experiment was to determine whether there was the same increase in the number of erythrocytes during experimental hyperleucocytosis as appeared clinically. The corpuscles in five cubic millimetres were counted at each examination.

Rabbit No. 9.

December 1. 3.45 P.M., red corpuscles, 6,075,200; white corpuscles, 8,800; white to red, 1-690. 4.15 P.M., two-tenths of a cubic centimetre of toxine solution injected into ear-vein.

December 2. 9.30 A.M., red corpuscles, 7,436,400; white corpuscles, 12,400; white to red, 1-600.

That is, there was an increase in the number of red corpuscles of a little more than twenty per cent., thus confirming the clinical observations. As stated, when considering the clinical examinations, I am unable to find any satisfactory explanation for this increase.

December 3. 10 A.M., rabbit moribund. Circulation so feeble that no blood could be obtained from ear.

December 4. 9 A.M., died during the previous afternoon.

At the autopsy the axillary glands were moderately enlarged and reddened; inguinal glands less so; mesenteric and retroperitoneal not at all. Suprarenals moderately reddened. Liver moderately congested, but showed no macroscopic necroses. Kidneys much congested. No fluid in thorax. Lungs moderately congested and edematous. Bone-marrow no redder than normal.

The results of my experiments coincide pretty closely with those obtained by Goldscheider and Jacob. They show that hyperleucocytosis occurs without local inflammation or exudate, and that, experimentally at any rate, hyperleucocytosis is preceded by hypoleucocytosis. The presence of an increased number of white cells in the lung capillaries during hypoleucocytosis, and to a less extent during hyperleucocytosis, is in direct contradiction to Löwit's theory of leucolysis. The absence of an increase of white cells in the liver and kidney during hypoleucocytosis and the persistence of an increased number in the lung capillaries during hyperleucocytosis prove that Schultz's supposition that hypoleucocytosis and hyperleucocytosis are merely the expression of a shifting of leucocytes is wrong.

As already noted, my results were the same as those obtained by Goldscheider and Jacob, except that I found rather fewer leucocytes in the lung capillaries during hyperleucocytosis than during hypoleucocytosis. This is a comparatively unimportant difference, however, as there are so many more there than in the normal lung that it is evident that at least only a part of the cells can have returned to the general circulation, and hence that the white cells in the general circulation during hyperleucocytosis must be largely

new accessions from the blood-forming organs; that is to say, my results corroborate the chemotaxic theory and seem explicable by it alone.

Everard concludes that there is a special diminution in the polynuclear cells with granular protoplasm during hypoleucocytosis, and that these same cells are very abundant during hyperleucocytosis. Löwit and Goldscheider and Jacob found almost exclusively mononuclear cells immediately after hypoleucocytosis, and that a decided preponderance of polynuclear cells arises during the course of the hyperleucocytosis. The proportions of the white corpuscles in the rabbit differ decidedly from those in man. The average of the differential counts made in the normal rabbits is as follows:

	Per cent.
Small Lymphocytes	43
Large Mononuclear and Transition Forms	8
Polynuclear Neutrophiles	11
Eosinophiles	38

That is, there are twice as many small mononuclear cells as in human blood, while the polynuclear neutrophiles, which make up the bulk of the white cells in human blood, here form but a small factor. The eosinophile cells, however, are very much more numerous, making more than a third of the total. They probably correspond to the polynuclear neutrophiles in the human blood and represent the oldest forms. Attention has already been called to their extreme abundance in the normal marrow. Two counts made during hypoleucocytosis (Rabbit 5) resulted as follows:

	Per cent.	
Small Lymphocytes	32.2	31.2
Large Mononuclear and Transition Forms	7.6	7.4
Polynuclear Neutrophiles	8.9	11.2
Eosinophiles	51.3	50.2

That is, there was a diminution in the small mononuclear cells and a proportionate increase in the polynuclear eosinophiles, which is directly the opposite of Everard's observation. No count was made at the beginning of hyperleucocytosis. One made at its height (Rabbit 8) resulted as follows:

	Per cent.
Small Lymphocytes	15.4
Large Mononuclear and Transition Forms	1.6
Polynuclear Neutrophiles	4.4
Eosinophiles	78.6

Another made during the subsidence of hyperleucocytosis was as follows:

	Per cent.
Small Lymphocytes	30.3
Large Mononuclear and Transition Forms	5.8
Polynuclear Neutrophiles	7.8
Eosinophiles	55.6

This agrees entirely with the results of other observers, and is undoubtedly the analogue of the increase in polynuclear neutrophiles in man. Goldscheider and Jacob lay much stress on this large proportion of eosinophiles, and consider that they are not entirely formed from the new mononuclear cells, but are partly old cells which were lying in the bone-marrow ready for extrusion, and have been brought into the circulation in increased numbers by chemotaxic action. A careful study of slips made from the fresh bone-marrow was very unsatisfactory. The impression obtained, however, was that the eosinophile cells were less plentiful in the marrow of hyperleucocytosis than in the normal. As mentioned above, the cut section of the marrow of hyperleucocytosis showed a diminution in the number of polynuclear cells. These observations, as far as they go, agree with Goldscheider and Jacob's position, that the increase in the eosinophiles is partly the result of the extrusion of the cells already formed in the marrow, and confirm the chemotaxic theory.

The gross pathological lesions observed in the five animals autopsied during hyperleucocytosis are of some interest. In one case there was no involvement of the lymph-glands. In all the others the axillary glands were enlarged and more or less congested, while the inguinal were affected in two, and the mesenteric and retroperitoneal in only one. The suprarenal capsules were involved in every case, being moderately reddened in three and much in two. The kidneys were enormously congested in four cases, and moder-

ately so in one. The liver was slightly congested in two, not at all in the remainder. No macroscopic necroses were observed, and none were found in the two cases examined microscopically. The thorax never contained fluid. More or less congestion of the lungs was present in every instance, however, and in two there was slight edema. The bone-marrow was redder than normal in three cases.

To sum up, the constant and most striking lesions were congestion of the kidneys, suprarenals, and lungs. More or less acute lymphademitis was usually present, and occasionally, edema of the lungs and reddening of the bone-marrow. Necrotic foci in the liver and fluid in the thoracic cavity, which were found so frequently by Wright [15] in guinea-pigs dead of experimental diphtheria, were not observed.

When my work was nearly completed, a paper was published by Gabritschewsky [16] entitled "Du Rôle des Leucocytes dans l'Infection Diphterique." He investigated the general leucocytosis of diphtheria in children and rabbits, and, in addition, studied the local leucocytosis and the action of the antitoxin serum on the progress of the leucocytosis. His conclusions regarding the general leucocytosis, based on his observations on fourteen children and on rabbits, are as follows: There is always a hyperleucocytosis in diphtheria, which is greatest in fatal cases, and which progressively diminishes during convalescence and after injections of antitoxin. A progressive hyperleucocytosis in diphtheria justifies a bad prognosis, and the analysis of the blood gives useful information regarding the value of treatment. He found that the number of white cells ordinarily varied between 11,450 and 25,000, and in fatal cases, between 29,500 and 51,000.

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XXII.

A REPORT ON THE BACTERIOLOGICAL INVESTIGATIONS OF AUTOPSIES.

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In this paper we report the results of the bacteriological study of a large number of autopsies, which have been performed at the Boston City Hospital and elsewhere, during a period of about two years prior to January 1, 1895.

This work has been carried on under the direction of Prof. W. T. Councilman, both in the pathological laboratory of the hospital, and in the Sears pathological laboratory of the Harvard Medical School.

METHODS OF STUDY.

As a rule, bacteriological examinations have been made from the various organs in every case which came to autopsy, the lung, liver, spleen, kidney, and blood of the heart being usually examined. In most cases culture methods only have been used to determine the presence and character of the bacteria in the organs, but in a few instances direct examinations by cover-slip preparations have been employed for this purpose, where this mode of examination has seemed to be adequate.

The culture medium used almost exclusively has been the coagulated blood-serum mixture of Löffler, prepared in tubes in the form of "slants." This medium is prepared by a modification, originated by Dr. F. B. Mallory, of the method generally followed, so that large quantities of it can be readily made up in a short time. The special points in this modified method consist in firmly coagulating the Löffler

blood-serum mixture (one part one per cent. grape-sugar bouillon and three parts beef blood-serum) in the ordinary dry-air sterilizer at 80° to 90° C., in the form of slant tubes, and then sterilizing it in the steam sterilizer during three successive days, as in the case of ordinary culture media.

The main difficulty to contend with is the formation of cavities and bubbles in the medium by too great or too rapid heating, so that the smooth surface of the "slant" is destroyed. This can be avoided, however, by securing a thorough and firm coagulation before placing the tubes in the steam sterilizer. Care should also be taken, in the coagulation by dry heat, not to allow the temperature to get above 90° C., or the same formation of cavities may take place. The culture medium thus prepared is opaque and of a creamy or brownish-white color, resembling in consistency coagulated egg albumin, and presents a smooth, almost perpendicular surface in the tube, upon which there is abundant space for the development of colonies.

The blood-serum used for this medium need not be collected with any special precautions to avoid contamination, and the amount of red-blood corpuscles in it is of little or no importance. The firm coagulation to which this mixture of blood-serum and bouillon is subjected does not seem to impair its nutritive qualities, as might be supposed, for one of us (Wright) has isolated the bacillus tuberculosis upon it directly from tubercular lesions, both in human and in experimental tuberculosis, — a fact which is an excellent proof of its efficiency for the cultivation of bacteria.

A further proof of its great value as a culture medium is afforded by the fact that the micrococcus lanceolatus (pneumococcus) and the streptococcus grow upon it much better and more readily than upon ordinary solid media.

The method of examination of organs by cultures consists in smearing a small amount of the tissue or blood to be examined, over the smooth surface of the culture medium in a tube, by means of a stiff, flattened platinum wire, under the usual precautions, and then placing the culture in the incubator for development. After twenty-four hours the colonies, which may have grown out upon the nutritive surface, are examined and their character determined.

If the organ is suspected of containing large numbers of bacteria, a second tube may be inoculated from the first one by touching the sterilized platinum wire to the infected surface of the first tube, and then rubbing the infected wire thoroughly over the surface of the second. Since usually only a comparatively few bacteria will be carried over by the platinum wire and distributed over the surface of the media in the second tube, it will be found that in this tube the colonies afterwards developing will be rarely so numerous as to be confluent. If desirable, a third tube may be similarly inoculated from the second; but this is rarely necessary, and, as a rule, one tube is sufficient.

The use of this coagulated blood-serum has greatly simplified the technique of the bacteriological examination of pathological material. It possesses so many advantages over the media usually employed that we have long abandoned the use of any other form of culture at autopsies. These advantages are:

(1) The fact that the micrococcus lanceolatus and the streptococcus grow on it much better and much more quickly than on ordinary media, such as agar-agar, while the bacillus diphtheriæ grows on it fully as well and quite as characteristically as on the blood-serum medium sterilized fractionally at low temperature, which costs so much labor to prepare.

(2) The ease with which the identification of the bacteria concerned in acute infections can be made; the microscopical examination of the original cultures made at the autopsy being in nearly all cases alone sufficient to establish the identity of the infecting organism, if this is considered in connection with the naked eye appearances of the colonies. For when cultivated on this medium the colonies of the pathogenic bacteria, which are ordinarily met with in human pathology, present much more characteristic appearances than when grown on agar-agar, so that to a great extent the diagnosis of the bacteria present may be made by macroscopic examination alone. Moreover, in cases of poly-infection, or infection with two or more species of bacteria, the estimation of the comparative numbers of the different species is greatly facilitated by the fact that all of the colonies are on the surface, where they can assume their characteristic form, and

not, as in agar-plate cultures, for instance, also in the depths of the medium, where their development is impeded, and such an estimation often difficult. We have been able repeatedly to identify with certainty three species of bacteria in a single tube, and to form a clear estimate of the relative number of each. The cultural peculiarities of the species above mentioned, when grown on this medium, are described elsewhere.

(3) A third advantage is the facility with which the medium can be prepared as compared with the agar-agar so generally employed, the preparation of which demands great patience and considerable experience.

(4) Finally, the quickness and simplicity of the method by which cultures on this medium are made at autopsies as compared with the usual methods of bacteriological examination by cultures. After a rather extensive experience in bacteriological work, we believe that the plate method and the method of Esmarch roll-tubes have no advantage over this simple method of successive "smears," when the object is to obtain discrete colonies from an infected organ, or to isolate bacteria in pure culture.

Of course, when it is desired to study colonies with a low power of the microscope, the smear method is not to be used; but since in the case of the colonies of the common pathogenic bacteria such study on the usual media gives little information beyond that to be obtained by the naked eye, the simpler methods seem to answer all the requirements in pathological work, especially if blood-serum be the medium employed.

METHODS OF BACTERIOLOGICAL DIAGNOSIS.

The main object which we have placed before ourselves in this work has been the recognition and the identification of the well-known bacteria concerned in the infectious processes which we have met with.

It is now clearly established that the important pathogenic bacteria which we have to deal with in the vast majority of cases of acute infectious disease in man are really few in number and comprise the staphylococcus pyogenes aureus, the streptococcus, the micrococcus lanceolatus, the bacillus

diphtheriæ, the typhoid bacillus, and the bacillus coli communis. We have, therefore, devoted our attention towards determining the presence and identity of these species, and have not occupied ourselves with the minute study of the various unknown bacteria which have appeared in our cultures from time to time, for either their presence could be accounted for by putrefactive processes, or there was no good evidence connecting them with the pathological conditions.

The chief characteristics of these half-dozen forms of pathogenic bacteria upon which we have based their recognition and identification are detailed in the following sections:

THE STAPHYLOCOCCUS PYOGENES AUREUS.

This organism grows on the coagulated blood-serum in the form of golden-yellow to orange-colored, slightly elevated, shining colonies, which may in two or three days attain a diameter of several millimetres. The great advantage in the use of this medium for the cultivation of this organism lies in the fact that the characteristic yellow color of its colonies appears immediately and not after several days, as in the case when agar-agar is used as a culture medium.

We have repeatedly recognized the presence of the staphylococcus pyogenes aureus when only a few of its colonies were present in the midst of hundreds of colonies of other bacteria, by means of this yellow color. In our work at autopsies we have not met with any other organism which produces this peculiar color, and so characteristic is it of the staphylococcus pyogenes aureus, when appearing in the cultures from an organ, that microscopic examination is hardly necessary.

The diagnosis of this organism, therefore, depends mainly upon the appearance of its colonies to the naked eye, and these, considered in connection with the results of the microscopical examination to determine the morphology of the constituted organisms, are amply sufficient for positiveness. The occurrence of the other variety of the staphylococcus pyogenes, namely, the albus, has been only very exceptionally observed in our studies.

THE STREPTOCOCCUS.

In agreement with the prevalent opinion among bacteriologists we make no distinction between the streptococcus found in erysipelas and that occurring in other conditions; but we consider that but one true species of streptococcus is concerned in human pathology, which, like the bacillus tuberculosis, is capable of inducing lesions of various forms, the form of lesion depending largely upon the tissue involved, and upon the duration of the infection.

The streptococcus grows on the coagulated blood-serum in the form of minute colonies, but more rapidly and better than upon the ordinary media. These colonies have no definite color, and when numerous are seen as fine grains or as a finely granular appearance on the surface of the medium. On microscopic examination these colonies are found to consist of cocci, in which the chain mode of proliferation may, or may not, be apparent, owing to the breaking up of the chains during the preparation of the cover-glass specimen for examination. Well-marked chains of streptococci can be usually demonstrated, however, in the fluid which is usually present at the bottom of the culture tube. This fluid constitutes essentially a bouillon culture, in which form of culture the organism finds the best conditions for chain formation, as is well known. The diagnosis of the streptococcus rests, then, upon the finding of definite chains of cocci in the culture by microscopical examination, and also upon the growth of these cocci in the form of minute colonies. The only bacterium with which the streptococcus may be confounded is the micrococcus lanceolatus.

THE MICROCOCCUS LANCEOLATUS.

The micrococcus lanceolatus also grows in the form of minute colonies; but these are usually more moist, flatter, more shining, and generally somewhat smaller than those of the streptococcus. The appearance of the colonies is quite characteristic. On microscopical examination the colonies of the micrococcus lanceolatus are seen to consist of pairs of small oval organisms often resembling pairs of small, short, oval, lancet-shaped or conical-ended bacilli. Some variation

in form and size is usually to be noted, and occasionally indications of capsule formation. In the fluid at the bottom of the culture tube longer and shorter chains may be found, which are sometimes very difficult to distinguish from chains of the streptococcus. By careful examination, however, it will commonly be found that the chain of the micrococcus lanceolatus is made up of pairs of oval organisms, while the streptococcus chain is composed of hemispherical cocci. Our diagnosis, therefore, of the micrococcus lanceolatus in cultures rests upon the minute size and the other appearances of its colonies, and the morphology of the organism itself. The pneumococcus may also be identified in the tissues by direct examination of stained preparations, by its morphology, and its characteristic capsule. It may be said also that the pathogenic effect of the bacterium exhibiting the characteristics above described has been repeatedly demonstrated on animals by the production of the characteristic septicemia after subcutaneous inoculation.

THE BACILLUS DIPHTHERIÆ.

The morphology of this organism when cultivated upon the blood-serum, prepared by the rapid method, is quite as characteristic as when this medium, prepared by the ordinary more difficult method, is employed.

We have found this medium especially adapted for determining the presence of the bacillus in the organs of cases dead of diphtheria, because its growth on the coagulated blood-serum is so much more luxuriant and certain than upon agar-agar. In fact, we consider that if agar-agar had been used instead of blood-serum in our work on diphtheria autopsies, we should have failed in many cases to have recognized the presence of the bacillus diphtheriæ, when that organism was found in very small numbers, as is most frequently the case in the viscera.

THE TYPHOID BACILLUS.

The colonies of the typhoid bacillus on the coagulated blood-serum are round, grayish-white disks, often several millimetres in diameter, and are essentially like the colonies

of the bacillus coli communis. For the identification of the typhoid bacillus we have not been satisfied with the microscopical examination of its growth on the serum, although this may give important information, if, in the fluid in the bottom of the culture tube, motile bacilli corresponding in morphology to the typhoid bacillus are seen, and the gross appearances of the colonies on the surface of the medium fulfil the requirements, but we have also applied the various tests which serve to distinguish this organism from the bacillus coli communis.

These tests are: the absence of gas-production in sugar agar or sugar gelatin cultures: the absence of any appreciable change in the litmus milk culture; the invisible growth on potato; the absence, or slight amount, of indol production in Dunham's pepton solution; active motility, and facility with which the locomotor organs, or flagella, may be stained by Löffler's method. Another distinguishing point, which we have found constant between the typhoid bacillus and the bacillus coli communis, is the less dense cloudiness of the bouillon culture of the typhoid organism.

THE BACILLUS COLI COMMUNIS.

The colonies of this organism on the coagulated blood-serum are round, shiny, moist, and of no definite color. They grow rapidly and may obtain a diameter of several millimetres. Our experience has shown us that when colonies of this character, made up of short bacilli of a morphology corresponding to that of the bacillus coli communis, appear in a culture from an organ, they are to be regarded as colonies of that organism almost certainly, and we have generally so considered them. We have satisfied ourselves that the bacillus growing in such colonies is the bacillus coli communis, by subjecting it in numerous cases to most of the various tests for the identification of that organism. These consist in the formation of gas in sugar gelatin, sugar agar-agar, or in glycerine agar-agar suspension cultures; in the reddening, and usually the coagulation also, of litmus milk cultures; in the moist slimy growth on potato: in the usual absence of motility; in the abundant production of indol in

Dunham's pepton solution; and in the difficulty of staining the flagella by Löffler's method. The bacillus coli communis has been met with very frequently in the organs of cases of all kinds, both infectious and otherwise. In fact, so frequent has been its occurrence in our cultures that its absence rather attracts attention, and we have ceased to attach much importance to its presence, in most cases at least. From our experience we are of the opinion that the significance of the presence of this bacillus in pathological processes has been greatly over-estimated.

For these reasons we have practically ignored the presence of the bacillus coli communis in reporting the results of our work. It should be understood, however, that in the vast majority of cases here reported it has been found in greater or less numbers along with the infecting bacteria in some or all of the organs.

ACUTE LOBAR PNEUMONIA.

It is now generally believed that the micrococcus lanceolatus is the only organism which has any etiological significance in acute lobar pneumonia.

The work of a host of investigators has proven this conclusively, and has further shown that the micrococcus lanceolatus is not only concerned in the causation of the disease, but that it is frequently the infecting organism in other acute inflammatory processes, both in the lung and elsewhere. This organism was first discovered in the salivary secretion by Sternberg and Pasteur in 1880, but it was not until 1886 that Fränkel and Weichselbaum demonstrated the occurrence of the same bacterium in the hepatized lung in the majority of cases, at least, of acute lobar pneumonia.

The early history of the micrococcus lanceolatus and of the bacteriology of acute lobar pneumonia are admirably presented by Welch, in a paper published two years ago.

Welch concludes, as the result of his study of the extensive literature on the subject of the bacteria concerned in acute lobar pneumonia and his own observations on fifty cases of the disease, that the micrococcus lanceolatus is undoubtedly its cause. Our own results are in harmony with this conclusion. While in a few instances we have found other

pathogenic bacteria in the solidified lung, as others have done, we do not attach any significance to them, for their presence can be usually explained on the ground of secondary infection.

We have never met with the bacillus of Friedlander, which Welch considers as "probably in no way concerned in the causation of genuine acute lobar pneumonia in man."

The occurrence of the micrococcus lanceolatus in the blood and internal organs in cases of acute lobar pneumonia has not received a great deal of attention in the literature. It has been found in the circulating blood, *intra vitam*, by Belfanti in 6 cases, and by Boulay in 2 cases. Post-mortem it has been found in the blood by Pernice and Alessi in 2 cases, and also by Netter (1 case) and Levy (1 case) in intra-uterine infection of the fetus with it. In the kidneys in cases of acute lobar pneumonia the micrococcus lanceolatus has been observed by Faulhaber in 29 cases and by Fränkel and Reiche in 22 out of 26 cases. The occurrence in the spleen has been noted by Prior (5 cases) and by Sée and Bordas. Welch, in his examination of 10 cases, found it in the spleen in 4, in the kidney in 1, and in the blood of the heart in 1 case.

In the course of our work bacteriological examinations of the hepatized lung in acute lobar pneumonia have been made in 42 cases, and the presence of micrococcus lanceolatus demonstrated in 38. Among these 42 cases there were 4 cases of the unresolved or organized form of acute lobar pneumonia, in 3 of which the micrococcus lanceolatus was found, in 1 accompanied by the staphylococcus pyogenes aureus, and in another accompanied by both that organism and the streptococcus.

The staphylococcus pyogenes aureus also accompanied the micrococcus lanceolatus in one of the cases of the ordinary form. With the exception of this case and the 2 cases of organized pneumonia just mentioned, in all of the positive cases the micrococcus lanceolatus has been present alone or associated with the bacillus coli communis, or similar non-significant organisms. In 2 of the 4 cases in which the micrococcus lanceolatus was not found, the negative result

may be ascribed to the large number of apparently unimportant bacteria which were present, and which may have prevented the development of the feebly growing micrococcus lanceolatus.

Of the 2 remaining negative cases, in one the staphylococcus pyogenes aureus and bacillus coli communis were found, and in the other the streptococcus, the bacillus diphtheriæ, and the staphylococcus pyogenes aureus.

In the first case there was coincident a purulent epididymitis with multiple abscesses in the kidneys, and in the second there had been empyema, thoracentesis, and general infection with the streptococcus. It seems probable that the abundant growth of the various bacteria found in these 2 cases in the lung may have obscured the recognition of the micrococcus lanceolatus or prevented its development in the culture, if present.

As to the occurrence of the micrococcus lanceolatus in the various organs in these cases of acute lobar pneumonia, our results may be summed up as follows:

The micrococcus lanceolatus has been found in the liver in 8 out of 26 cases examined; in the spleen in 6 out of 26 cases; in the kidney in 11 out of 26 cases; and in the blood of the heart in 3 out of 11 cases. It is apparent, therefore, that there is more of a general invasion of the blood by this organism than is generally supposed.

PLEURITIDES.

The most extensive investigations of the bacteria concerned in exudative pleuritis are those of Levy, Netter, Koplick, Jakouski, Prince Ludwig Ferdinand, and Prudden. The results obtained by these bacteriologists and others have shown that there is no one species of bacteria constantly associated with this condition, but that any of the common pathogenic bacteria and others may be present. From the work of Prince Ludwig Ferdinand and of Prudden, it would appear that cases in which the micrococcus lanceolatus is found have a better prognosis than others.

Of 13 cases of fibrinous pleuritis accompanying acute lobar pneumonia the micrococcus lanceolatus was found in 12,

in practically pure cultures in nearly every case, the contaminating organism being usually the bacillus coli communis. In the single negative case various unknown bacteria were present. In all of 4 cases of purulent pleuritis coincident with acute lobar pneumonia the micrococcus lanceolatus has been present, in 2 cases in company with the staphylococcus pyogenes aureus. Of 3 additional cases of fibrinous pleuritis, not accompanying acute lobar pneumonia, the micrococcus lanceolatus was found in 2 (in 1 along with the staphylococcus pyogenes aureus) and the streptococcus in the third case.

Also, in 2 out of 3 additional cases of purulent pleuritis, not associated with acute lobar pneumonia, the micrococcus lanceolatus was present, in 1 case with the streptococcus, and in the other with various unknown bacteria. In the third case the staphylococcus pyogenes aureus was found (culture taken a few hours before death). This was a case of purulent broncho-pneumonia and general infection with the staphylococcus pyogenes aureus.

ACUTE PERICARDITIS.

The bacteriological examinations of the exudate in acute pericarditis recorded in the literature are not numerous.

Weichselbaum has found the micrococcus lanceolatus in several cases; Bainti, the same organism in 2 of 3 cases; Steinberg in 1 case; and Thue in 4 out of 5 cases. Barbacci, in the examination of 3 cases found the staphylococcus pyogenes aureus in 1, and the micrococcus lanceolatus in 2 cases, while Foureur in 1 case found the streptococcus.

The occurrence of other bacteria in pericarditis has also been reported by Ernst (tubercular pericarditis), and by Paviot.

In 7 out of 10 cases of acute pericarditis, 7 of which accompanied acute lobar pneumonia, the micrococcus lanceolatus has been present. Of the 3 negative cases, in 2 only the bacillus coli communis was found, while the other case was sterile. In 1 additional case of acute pericarditis in which no culture was taken from the pericardial exudate, a streptococcus septicemia was demonstrated, so

that it is fair to conclude that the pericarditis was due to that organism.

It may also be mentioned that a case of hemorrhagic pericarditis after abortion was negative for specific bacteria.

PURULENT LEPTO-MENINGITIS.

The extensive literature on the subject of the bacteria associated with purulent lepto-meningitis has recently been summarized by Flexner and Barker in their interesting study of epidemic cerebro-spinal meningitis. According to these writers, the micrococcus lanceolatus has been found in the great majority of the numerous cases recorded in the literature, while the streptococcus, the staphylococcus pyogenes aureus, and other bacteria have been met with in but a small proportion of the cases.

This very frequent occurrence of the micrococcus lanceolatus in purulent inflammation of the pia mater has been observed in the cases examined by Flexner and Barker, as well as in those here reported.

In 8 of 11 cases of purulent lepto-meningitis and in 1 case of the sub-acute form the micrococcus lanceolatus has been present. The staphylococcus pyogenes aureus accompanied the micrococcus lanceolatus in 1 of the 8 cases of purulent meningitis.

Of the remaining 3 cases of purulent meningitis, in 1 the streptococcus was found and 2 were negative; but 1 of these negative cases was probably due also to a streptococcus infection proceeding from an otitis media, in the pus of which that organism was present.

Two of the micrococcus lanceolatus cases were secondary to fracture of the base of the skull, the fracture going through the petrous portion of the temporal bone, and thus, by presumably placing the brain cavity in communication with the pharynx, through the Eustachian tube, an infectionatrium for the micrococcus lanceolatus, so common an inhabitant of the throat, was provided. Acute lobar pneumonia was coincident in two of the micrococcus lanceolatus cases, and in the negative case. Otitis media was also coincident in one of the same cases.

ACUTE ENDOCARDITIS.

The evidence in favor of the bacterial origin of acute endocarditis is both experimental and pathological.

The possibility of inducing an experimental acute endocarditis, due to bacteria, was first suggested by Rosenbach, in 1878, who showed that by injuring the heart-valves of an animal by means of a sound passed into the carotid, in certain cases vegetations were produced on the valve in which microörganisms could be demonstrated. These microorganisms were carried into the circulation accidentally by the operation, and Rosenbach considered that a certain proportion of the heart-lesions resulting was due to their presence and was independent of the mechanical injury produced by the sound. These experiments of Rosenbach opened the way for the later work of Wyssokowitsch and Orth in 1885, who proved that by the injection of the pyogenic cocci into the circulation of animals, after previous mechanical injury to the valves of the heart by Rosenbach's method, a true acute endocarditis could be induced. These investigators maintained that the previous lesion of the valves was necessary, for the mere injection of the pyogenic cocci into the circulation was not followed by the appearance of valvular vegetations, nor did such vegetations appear after the mechanical injury of the valves, if no bacteria were introduced into the blood. The experimental production of acute endocarditis has also been studied by Netter, Ribbert, Fränkel and Sänger, Prudden, Weichselbaum, Stern and Hirschler, and Viti. The combined results of the work of these writers have shown that true endocarditis can readily be produced in animals by the intravenous injection of any of a number of species of bacteria, provided that there is some previous lesion of the heart-valves, although the necessity of this previous lesion is denied by Netter and Ribbert.

The pathological evidence of the infectious nature of acute endocarditis is also convincing. In 1885 Wyssokowitsch reported finding the staphylococcus pyogenes aureus in a case of ulcerative endocarditis, but failed to find any bacteria in 11 cases of the verrucose form. In the same year Weichselbaum published the results of his study of 1 case of the

verrucose form and 3 cases of the ulcerative form, in all of which he found either the staphylococcus pyogenes aureus or the streptococcus, or both. The first extensive study, however, of the etiology of acute endocarditis in man is that of Fränkel and Sänger, published in 1886. These investigators studied 11 cases of the verrucose form and 1 case of the ulcerative form, and found in 9 cases bacteria in the cardiac lesions, the staphylococcus pyogenes aureus being present in 6. This publication was important because it was a strong confirmation of the view, which is now generally held, that all forms of acute endocarditis are to be regarded as of infectious origin. An important work also is that of Netter, who observed the micrococcus lanceolatus in 7 or 9 cases of acute endocarditis associated with acute lobar penumonia.

Weichselbaum has, however, contributed more to our knowledge of this disease than any other single investigator. From a study of 15 cases of ulcerative endocarditis, 13 cases of verrucose endocarditis, and 1 case of endocarditis of the left auricle, Weichselbaum concluded that there is no essential difference between the various forms of endocarditis, either histologically or bacteriologically ; that no one species of bacteria is concerned in the production of the disease ; that in cases in which no bacteria are found, the bacteria have died, or there is some defect in the method of examination for their presence ; and that chronic endocarditis is the result of the acute form of the disease. Of the 29 cases of Weichselbaum, the micrococcus lanceolatus was found in 7 (6 of the ulcerative form and 1 of the verrucose form) ; the streptococcus in 6 (4 ulcerative and 2 verrucose) ; the staphylococcus pyogenes aureus in 2 (verrucose) ; and in 6 cases various unusual bacteria. In 8 cases the examination for the presence of bacteria was negative.

Other observations of bacteria in acute endocarditis are those of Banti (20 cases), Viti (8 cases), Girode (6 cases), Senger, Lanceraux, Stern and Hirschler, Meyer, Vinay, Steinberg, Malvolz, Tombolan and Fava, Perret and Rodet, Gilbert and Lion, Paulus, Lanceraux and Besançon, Oulmont and Barbier, Lafitte, Josser and Roux, Barbacci, Glusincki, Leyden, Howard, and Councilman. The cases

studied by these writers prove conclusively that a great variety of bacteria are to be found associated with the acute forms of the disease, in which not only the ordinary pyogenic cocci, the micrococcus lanceolatus, and other less known species, may be present, but also the gonococcus (Councilman and others) and the bacillus diphtheriæ (Howard) as well.

In 10 cases of acute endocarditis examinations of the valvular vegetations or ulcerating tissue of the heart have been made, and in 7 of the 10 cases the micrococcus lanceolatus has been found, in 1 case accompanied by the bacillus diphtheriæ. Of the 3 remaining cases in which the micrococcus lanceolatus was not found, the staphylococcus pyogenes aureus was present in one, the streptococcus and the staphylococcus pyogenes aureus in the second, and unknown bacteria in the third case. In all, 19 cases of acute endocarditis, 6 or 7 of which were of the ulcerative form, have been studied with reference to the occurrence of bacteria in the internal organs. The micrococcus lanceolatus has been found in the heart-blood, or in some or all of the parenchymatous viscera, in 7 cases. Combining the results of these cultures with the results of the examination of the heart lesions mentioned above, it may be said that evidence of the association of the micrococcus lanceolatus with acute endocarditis has been obtained in 9 out of the 19 cases. Of the remaining cases, in 2 there was a well-marked general infection with the staphylococcus pyogenes aureus; in 2 others, both in puerperal females, a general infection with the streptococcus; and in 6 no definite conclusion was to be drawn from the results of the cultures. The staphylococcus pyogenes aureus was also present with the streptococcus in the blood of the liver as well as in the valvular vegetations in one of these puerperal cases. It is worthy of note that in a third puerperal case, with acute endocarditis, the micrococcus lanceolatus was found in the blood of the heart and in the embolic pneumonia, while the streptococcus was found in the uterus and kidney.

GENERAL INFECTIONS WITH THE MICROCOCCUS LANCEOLATUS.

In addition to the foregoing cases of general infection with the micrococcus lanceolatus in acute lobar pneumonia and acute endocarditis, a number of other cases have been met with in which this organism has been found in some or all of the large viscera. These cases are essentially as follows:

Case I. — Acute fibrinous pericarditis with effusion. Acute fibrinous pleuritis, acute splenic tumor. Acute degeneration of liver and kidneys. Micrococcus lanceolatus in pericardium, lung, liver, and kidney. Spleen sterile.

Case II. — Ulcus ventriculi; abscess of adjacent fat tissue with perforation into peritoneal cavity. Fibrinous peritonitis and pleuritis. Micrococcus lanceolatus in fibrinous exudate on peritoneum and pleura, in spleen, kidney, and wall of heart. Bacillus coli communis in liver and in some of the other organs also.

Case III. — Hysterectomy. Occlusion of ureters by ligature, leakage of urine into peritoneal cavity, congestion of lungs, acute splenic tumor. Micrococcus lanceolatus in peritoneum and spleen. Blood of heart negative. Various bacteria were also present in peritoneum.

Case IV. — Acute adenitis of inguinal and peritoneal lymph-glands, acute splenic tumor, edema and congestion of lungs. No infection-atrium demonstrated. Micrococcus lanceolatus in inguinal lymph-gland, kidney, and liver. Spleen and femoral lymph-gland sterile. Bacillus coli communis in liver.

Case V. — Fracture of base of skull extending through the petrous portion of the left temporal bone. Acute purulent otitis media (left), purulent leptomeningitis, thrombosis of left lateral sinus, acute splenic tumor, acute degeneration of liver and kidneys. The otitis was apparently subsequent to the fracture. Micrococcus lanceolatus in pus of internal ear and of meninges, blood of heart, liver, spleen, kidney, and lung. Staphylococcus pyogenes aureus also in lung. As elsewhere pointed out, the infection-atrium in this case was most probably the internal ear.

Case VI. — Subacute diffuse nephritis (large white kidney), anasarca, ascites, hydrothorax, subacute peritonitis.

Micrococcus lanceolatus in peritoneal exudate, spleen, kidney, liver, and blood of heart.

Case VII. — Disseminated general sarcomatosis. Broncho-pneumonia. Micrococcus lanceolatus in broncho-pneumonia liver, kidney, and blood of heart. Staphylococcus pyogenes aureus also in kidney. Spleen sterile.

Case VIII. — Child, aged two and one-half days. Acute pneumonia of both lungs. Hemorrhages beneath dura and peritoneum. Micrococcus lanceolatus by cover-slips (direct examination) in lung, liver, spleen, kidney, and blood of heart. There was no evidence of infection from mother.

Additional cases of general infection with the micrococcus lanceolatus are considered under puerperal infection, diphtheria, and scarlet fever.

MISCELLANEOUS GENERAL INFECTIONS WITH THE STREPTOCOCCUS.

In this group we have collected a variety of cases in which the bacteriological examination showed more or less of a general invasion of the viscera by the streptococcus. Some of these cases have been of a surgical character, but a number of others have been cases in which there was little or no reason for suspecting any pyogenic infection, either clinically or at the autopsy. With the exception of a single case, none of the cases with general infection with the streptococcus in diphtheria and scarlet fever are included in this group, but are considered elsewhere in connection with these diseases. The exceptional case was one of diphtheria and scarlet fever in which the streptococcus septicemia was apparently of surgical origin, and on that account it is included here.

Case I. — General arterio-sclerosis, hypertrophy and dilatation of the heart, thrombosis of right auricular appendix, multiple emboli of lungs with infarction, arterio-sclerotic kidney and liver, edema, ascites, and hydrothorax. Streptococcus in liver, kidney, spleen, infarction of lung and blood of heart.

Case II. — Disseminated tuberculosis of lungs. Tuberculosis of epididymus, seminal vesicles, and kidney, acute splenic tumor. Streptococcus in spleen and lungs; in lung,

also, bacillus diphtheriæ and staphylococcus pyogenes aureus. Kidney and liver sterile.

Case III. — General hematogenous miliary tuberculosis. Primary tubercular ulcer of intestine, tuberculosis of mesenteric glands and thoracic duct. Streptococcus in spleen, liver, kidney, lung, blood of heart, and in superficial vein.

Case IV. — Cancer of rectum. Streptococcus in kidney, lung, and blood of heart.

Case V. — Subcutaneous injections of bouillon cultures of the streptococcus derived from erysipelas, in a case of sarcoma of the thigh. Death after about ten days. At the autopsy there was found edema and congestion of the lungs, hemorrhages in parietal and visceral pleuræ, enlargement and congestion of the inguinal glands on the side of the tumor, and of the posterior mesenteric and gastro-hepatic lymph-glands. Streptococcus in tumor, inguinal gland, spleen, liver, and blood of heart. Bacillus pyocyaneus in tumor.

Case VI. — Broncho-pneumonia, purulent bronchitis, mucopurulent cystitis. Streptococcus in spleen, liver, kidney, and blood of heart.

Case VII. — Operation for congenital hernia, followed by abscess formation. Diphtheria. This case is included among the diphtheria cases. Streptococcus in liver, spleen, lung, and kidney. Bacillus diphtheriæ in throat, lung, and spleen. It seems probable that in this case the infection-atrium for the streptococcus was the operation wound.

Case VIII. — Acute lobar empyema, thoracentesis, croupous pneumonia, acute splenic tumor, fatty degeneration of liver, chronic diffuse nephritis. Larynx and trachea not examined. No suspicion of diphtheria. Streptococcus in kidney, spleen, liver, lung, and blood of heart. Bacillus diphtheriæ and staphylococcus pyogenes aureus also in lung.

Case IX. — Operation for tuberculosis of hip-joint. Acute peri- and endocarditis. Chronic endocarditis with general chronic passive congestion. Streptococcus in operation wound, liver, spleen, kidney, and heart muscle. Staphylococcus pyogenes aureus also in the operation wound.

Case X. — Ulcers and cellulitis of leg, purulent arthritis of knee. Broncho-pneumonia. Fatty degeneration of liver.

Acute splenic tumor. Acute degeneration of kidney. Diphtheritic ulceration of large intestine. Streptococcus in knee-joint, kidney, spleen, blood of heart, broncho-pneumonia, and possibly in liver. It is possible that there was a general poly-infection to a certain extent with both the streptococcus and the micrococcus lanceolatus, but this was not determined accurately.

Case XI. — Operation wound, removal of vermiform appendix, acute purulent peritonitis, broncho-pneumonia, acute parenchymatous degeneration of liver and kidneys. Streptococcus in peritoneal pus, liver, and kidney.

PUERPERAL INFECTION.

The present state of our knowledge on the subject of puerperal infection has been recently reviewed by Williams, who concludes "that various, very diverse micro-organisms, especially the streptococcus and the staphylococcus," may be concerned in this condition, but "that most of the fatal cases of puerperal disease have been due to infection with the streptococcus." Among the first observations of the occurrence of the streptococcus in puerperal infection was that of Orth, in 1873, who described and figured a spherical organism, growing in chains in the purulent exudation of puerperal peritonitis. The first to cultivate this organism from autopsies on puerperal cases was Pasteur, in 1880, and since that time its frequent occurrence in puerperal infection has been demonstrated by Winkel, Pfannenstiel, Czerniewski, and others.

In this group of cases we have included all those cases of infection which seemed to have been of puerperal origin. It will be noted that some of them are well-marked poly-infections.

Case I. — Typical diphtheritic endometritis and vaginitis, abscesses in lungs, acute fibrinous pleuritis, acute splenic tumor. Streptococcus in pus of uterine lymphatics, blood of heart, and spleen. Streptococcus, staphylococcus pyogenes aureus, and micrococcus lanceolatus in lung (abscesses).

Case II. — Diphtheritic endometritis and vaginitis, abscesses in lung, acute pleuritis and parotitis, acute splenic

tumor. Criminal abortion at third month. Staphylococcus pyogenes aureus in parotid gland, blood of heart, and lung; in the lung also streptococcus. Liver and spleen sterile. Various unknown bacteria in uterus.

Case III. — Fever after childbirth, with intense jaundice. Acute nephritis. Streptococcus in liver, spleen, kidney, and blood of heart.

Case IV. — Abortion and septic endometritis. Streptococcus in uterus, pus of Fallopian tube, and in spleen. Liver and kidney negative.

Case V. — Puerperal fever. Streptococcus in uterus, thrombosed uterine vein and kidney. Other organs not examined.

Case VI. — Acute puerperal diphtheritic vaginitis, thrombosis of vaginal veins and uterine lymph-vessels, purulent peritonitis, acute pleuritis, congestion and edema of lungs, acute parenchymatous degeneration of liver and kidney. Congenital defect in intra-ventricular septum, patency of foramen ovale, hypoplasia of aorta; chronic passive congestion of lungs. Streptococcus in abdominal pus, pleural exudate and blood of heart. Staphylococcus pyogenes aureus in spleen. Bacillus coli communis in uterine lymph-sinus, vaginal veins, kidney, and in some other situations.

Case VII. — Hemorrhagic pericarditis, uterine hemorrhage, abortion in progress. Bacillus coli communis in body of uterus, kidney, blood of heart, and spleen. Liver sterile. Pericardium negative.

Case VIII. — Puerperal septicemia, partially retained placenta, acute splenic tumor, acute parenchymatous degeneration of liver and kidneys. Streptococcus in kidney. Bacillus coli communis in uterus, liver, spleen, kidney, and blood of heart.

Case IX. — Acute lobar pneumonia following childbirth. Streptococcus in uterus and kidney. Micrococcus lanceolatus in lung and pleural exudate. Bacillus coli communis in all organs.

Case X. — Hypertrophy of uterus after early abortion. Endometritis, acute endocarditis, hemorrhagic infarction of lungs with embolic pneumonia, acute splenic tumor. Micrococcus lanceolatus in blood of heart and lung. Streptococ-

cus in uterus and kidney. Bacillus coli communis in liver and in other organs.

Case XI. — Acute endometritis. Post-partum hypertrophy of uterus, acute endocarditis, septic infarction of spleen and kidneys, embolism of branches of superior mesenteric artery, subacute nephritis, slight chronic passive congestion of lung, liver, and spleen. Streptococcus in endometrium, liver, kidney, spleen infarction. Streptococcus and staphylococcus pyogenes aureus in heart-lesion and blood of liver vein. Spleen sterile.

Case XII. — Clinical diagnosis: puerperal eclampsia. Post-partum hypertrophy of uterus, laceration of cervix with hemorrhage into ligamentum latum. Hydrothorax, hydropericardium, edema of lungs, acute splenic tumor. Micrococcus lanceolatus in endometrium, liver, lung, spleen, and kidney.

The streptococcus has been found in one or more of the internal organs in nine of these twelve cases. This result is in harmony with the generally accepted view that this organism is by far the most common cause of puerperal infection.

ERYSIPELAS.

Case I. — Erysipelas of face, pulmonary apoplexy, acute splenic tumor. Old tuberculosis of apices of lungs, scattered foci of tuberculosis in the upper lobe of right lung, acute tubercular pleuritis left side. Streptococcus in tissue of face. Staphylococcus pyogenes aureus also in tissue of face and in spleen, kidney, lung, and blood of heart. Liver negative.

Case II. — Erysipelas of face and neck. Acute nephritis. Cultures failed to demonstrate any specific infection.

Case III. — Erysipelas of face and scalp, congestion of lungs, acute splenic tumor. Steptococcus and other organisms in scalp. Liver and spleen sterile. Various bacteria in blood of heart and lateral sinus. Staphylococcus pyogenes aureus in lung.

Case IV. — Erysipelas of face and scalp. Diffuse visceral syphilis. Streptococcus in blood of heart, spleen, and lung. Bacillus coli communis in kidney.

Case V. — Erysipelas of face. Contusion of forehead and buttock. Fatty degeneration and purulent infiltration of muscles of buttock. Streptococcus in muscles of buttock, spleen, kidney, liver, lung, and blood of heart.

Case VI. — Erysipelas of leg; small ulcer on posterior surface of leg. Inguinal glands on affected side enlarged and reddened, and in the adjacent tissue small purulent foci. Hypostatic congestion of lungs, acute splenic tumor. Streptococcus in the deep subcutaneous tissue of leg and inguinal gland. Micrococcus lanceolatus in lung. Bacillus coli communis in kidney and blood of heart.

Case VII. — Erysipelas of leg. Broncho-pneumonia. Tissues of leg and inguinal gland sterile. Streptococcus in liver. Staphylococcus pyogenes aureus in lung, kidney, and spleen. Blood of heart sterile.

It will be seen that the streptococcus has been found associated with six of these cases, while in two there was a general infection with the staphylococcus pyogenes aureus in addition. We consider the general infection with the latter organism as a secondary one, following on the erysipelas, which was induced by the streptococcus. The failure to demonstrate the streptococcus in the local lesion in one of the cases in which that was examined, is not surprising, as it is well known that it does not long survive in the tissue after it has produced its effects.

We have examined four cases of extensive superficial burns which we will consider in one group. The bacteriological results are as follows:

Case I. — Streptococcus in spleen, kidney, and blood of heart. Liver and mesenteric gland sterile.

Case II. — Streptococcus in suppurating tracheal gland, liver, spleen, and kidney.

Case III. — Bacillus coli communis in spleen, kidney, and lung. Liver and blood of heart sterile.

Case IV. — Various unknown bacteria found in the various organs.

GENERAL INFECTIONS WITH THE STAPHYLOCOCCUS PYOGENES AUREUS.

In this group we consider those cases in which the bacteriological examination showed a general mono-infection essentially with the staphylococcus pyogenes aureus. One of the cases of diphtheria is included here because the general infection with the staphylococcus seemed to have its origin in an operation wound of the hip, rather than the throat. We also report here more in detail the two cases of endocarditis with staphylococcus septicemia.

Case I. — Infection-atrium a decubitus over the scapula. Edema and hypostatic congestion of lungs. Acute splenic tumor. Arterio-sclerosis of aorta and heart hypertrophy. Staphylococcus pyogenes aureus in spleen, lung, liver, and kidney, and cerebral meninges. Streptococcus and micrococcus lanceolatus in lung.

Case II. — Oöphorectomy for cancer. Metastases in retroperitoneal lymph-glands, omentum, and liver. Circumscribed peritonitis. Acute splenic tumor. Chronic diffuse nephritis. Chronic pleuritis with effusion. Hypostatic congestion and edema of lungs. Staphylococcus pyogenes aureus in kidney and blood of heart.

Case III. — Laparotomy for chronic salpingitis, slight peritonitis. Soft spleen. Staphylococcus pyogenes aureus in the peritoneum, spleen, and blood of heart.

Case IV. — Operation for congenital dislocation of hip. Abscess about hip-joint extending around ileum, beneath peritoneum, up to the kidney. Secondary abscess in left shoulder, kidney, and lung. Diphtheria. Staphylococcus pyogenes aureus in primary abscess, kidney, spleen, liver, and lung. Bacillus diphtheriæ in air-passage and lung.

Case V. — General infection with actinomyces. The cultures showed a general infection with staphylococcus pyogenes aureus also. This case is fully reported elsewhere by Dr. Mallory.

Case VI. — Carbuncle of upper lip, metastatic abscesses of lung and kidney. Acute endocarditis, acute splenic tumor, acute hyperplasia of cervical and mesenteric lymph-glands. Acute parenchymatous degeneration of liver and kidneys.

Acute degeneration of heart-muscle. Staphylococcus pyogenes aureus in carbuncle of lip, kidney, spleen, lung, pericardium, and blood of heart.

Case VII. — Pyo-pneumothorax, perforation of diaphragm. Extensive purulent excavation of liver, general peritonitis. Pleural effusion (left side), fatty degeneration of liver. Staphylococcus pyogenes aureus in remnants of lung, liver substance, liver pus, and in kidney.

Case VIII. — Acute ulcerative endocarditis, acute pericarditis, infarction of kidney, unresolved pneumonia, empyema, chronic passive congestion. General hypertrophy and dilatation of heart. General edema of connective tissue. Staphylococcus pyogenes aureus in heart-lesion, spleen, kidney, kidney infarction, liver, and blood of heart.

Case IX. — Purulent broncho-pneumonia, empyema of one side. broncho-pneumonia and acute pleuritis of other side. Multiple miliary abscesses in kidney. Staphylococcus pyogenes aureus in lung (purulent broncho-pneumonia), liver, and spleen. Kidney abscesses sterile.

Case X. — Acute alcoholismus. Congestion of lung and stomach. Acute splenic tumor. Acute parenchymatous degeneration of liver. Staphylococcus pyogenes aureus in lung, liver, and kidney. Spleen sterile. Pneumococcus also in lung. A mouse inoculated with an impure culture of the pneumococcus found in the lung died in forty-eight hours with typical encapsulated diplococci in the blood.

ANOMALOUS GENERAL POLY-INFECTIONS.

Under the head of anomalous general poly-infection we group certain of the cases in which the staphylococcus pyogenes aureus and the streptococcus, or either of these organisms and the micrococcus lanceolatus, have been found together in the same case in some of the internal organs other than in the lung. The poly-infections of this character which have been observed in diphtheria, scarlet fever, erysipelas, and puerperal cases are not included in this group, but are considered elsewhere.

Case I. — Cirrhosis of liver, chronic interstitial nephritis, dilatation of right ventricle of heart, fibrous myocarditis, general chronic passive congestion, ascites and hydrothorax.

Staphylococcus pyogenes aureus in liver and kidney. Streptococcus and staphylococcus pyogenes aureus in spleen. No cultures from lung and heart.

Case II. — Extreme hemoglobinemia, hemoglobinuria, icterus, acute splenic tumor, acute fatty degeneration of heart-muscle. Micrococcus lanceolatus and staphylococcus pyogenes aureus in kidney. Micrococcus lanceolatus and streptococcus in lung. Streptococcus in liver. Spleen sterile. Unknown bacteria in blood of femoral vein.

Case III. — Meningitis purulenta, secondary to otitis media. Acute splenic tumor. Streptococcus in pus of otitis and in liver. Staphylococcus pyogenes aureus in kidney and lung. Bacillus coli communis in blood of heart. Spleen sterile.

Case IV. — Acute lobar pneumonia, acute pleuritis, acute splenic tumor, urinary fistula, abscess of kidney, acute degeneration of kidneys, cirrhosis of liver, arterio-sclerosis of aorta and coronary arteries, atrophy of pancreas. Streptococcus in liver, staphylococcus albus (?) in kidney, large coccus in spleen. Various bacteria in lung.

Case V. — Chronic diffuse nephritis, fatty kidney. Edema of brain and lungs, arterio-sclerosis of coronary arteries, muco-purulent bronchitis, acute splenic tumor. Micrococcus lanceolatus and streptococcus in liver. Micrococcus lanceolatus in kidney and spleen. Blood of heart negative. A mouse inoculated with the mixed culture from the liver died in about forty-eight hours with a streptococcus septicemia, as shown by cultures from the organs.

Case VI. — Chronic coxitis with suppurating foci, peritonitis with serous exudation, fibrinous focal pneumonia left lung, subacute nephritis and general amyloid infiltration. Micrococcus lanceolatus in peritoneal exudate, lung, and kidney. Streptococcus in liver, spleen, and also in kidney.

Case VII. — Bronchiectatic abscesses in lungs, organized pneumonia, subacute pleuritis with pus formation. Acute splenic tumor. Subacute nephritis. Micrococcus lanceolatus and staphylococcus pyogenes aureus in lung, pleural-pus, and kidney. Staphylococcus pyogenes aureus in liver. Spleen sterile.

Case VIII. — Acute lobar pneumonia, acute fibrinous

pleuritis. Acute splenic tumor. Micrococcus lanceolatus and staphylococcus pyogenes aureus in lung, liver, kidney, and blood of heart. Micrococcus lanceolatus in pleura, spleen, and blood of femoral vein.

DIPHTHERIA.

Up to the time of the well-known work of Frosch, it was generally believed that the *bacillus diphtheriæ* never invaded the internal organs, but was only to be found in the local inflammatory lesions. In ten out of fifteen autopsies on this disease, this investigator, by using large amounts of material for each culture, could demonstrate the presence of the bacillus either in the blood or in some of the organs. One of the most frequent situations in which it was found was in the pneumonic areas of the lungs. Previous to the work of Frosch but a very few instances are on record in which the bacillus has been met with in these situations. One of these is its occurrence in the spleen in a case reported by Kolisko and Paltauf. In the cervical lymph-glands it has been observed by Schmorl in seven out of ten cases. Recently Booker obtained it in cultures from the spleen, submaxillary gland, lung, and blood of the heart in a case of diphtheria, and Kutcher observed it once in the liver and once in the kidney.

In the broncho-pneumonia of diphtheria the presence of the bacillus diphtheria was noted by Thaon in 1885, but its occurrence in this common complication of the disease does not seem to have received much attention until the last few years, during which time Johnston found it in a single case, Strelitz in 1 case out of 8, Flexner in 1 of 2 cases, Mosny in 1 of 3, Kutcher in 8 out of 9, while the frequency of its occurrence in the cases examined by Frosch has been already mentioned. Of other bacteria associated with the broncho-pneumonia of diphtheria, the work of Thaon, A. Fränkel, Prudden and Northrup, Mosny and Netter, shows that the streptococcus is to be met with in the great majority of cases. In the 2 cases reported by Flexner, however, the micrococcus lanceolatus was present, and of the 8 cases of Strelitz', the same organism was present in 5, in 3 of which it was accompanied by the pyogenic cocci.

The occurrence of a poly-infection in certain cases of diphtheria with both the specific bacillus and the streptococcus has been insisted upon by Barbier. This writer holds that there exist two forms of the disease, one a pure mono-infection with the bacillus diphtheriæ, the other a poly-infection with that organism and the streptococcus, or various (doubtful) varieties of it. This conclusion is based mainly upon the results of the bacteriological examination of the throat during life. The post-mortem demonstration, however, of the presence of pyogenic cocci in the internal organs has apparently been made in but a few cases.

Geneserich, in an examination of twenty-five autopsies on cases of diphtheria of different clinical types, found among these, four cases of invasion of the blood and internal organs with the streptococcus, while in many cases a general infection with the staphylococcus pyogenes albus was observed.

Bacteriological examinations of the various internal organs have been made by us in 31 cases of diphtheria, in 5 of which scarlet fever was coincident, and in another case typhoid fever. The bacillus diphtheriæ has been found by culture in the lung in 30 out of 31 cases examined, in the liver in 9 out of 29 cases, in the kidney in 6 out of 31, in the spleen in 5 out of 31, in the blood of the heart in 5 out of 26, in the mesenteric lymph-glands in 7 out of 16, in the cervical lymph-glands in 4 out of 9, in the bronchial lymph-glands in 2 out of 3, in the brain tissue in 2 out of 5 cases, and in the mucous membrane of the stomach in 3 cases. In 21 of the 31 cases, or in nearly 70 per cent., there has been found a more or less general invasion of the internal organs by the pyogenic bacteria, of which the streptococcus was the one most frequently met with.

The streptococcus was present in one or more of the large viscera (exclusive of the lungs), or in the blood of the heart, or in both, in 20 cases. In 5 of these cases, in addition to the streptococcus in certain organs, the staphylococcus pyogenes aureus or the micrococcus lanceolatus, or both, have been found also in some of the organs of the same case.

Among the streptococcus septicemias was the case of diphtheria and typhoid fever reported by Morse, in which

the typhoid bacillus was also found in the various viscera. The only case of pyogenic infection in diphtheria in which the streptococcus was not concerned, but in which the staphylococcus pyogenes aureus was cultivated from all the large viscera, was one with a recent operation wound about the hip-joint, which should be regarded as the probable infection-atrium for the staphylococcus. Because of its unusual features, this case should be excluded from consideration as an ordinary case of poly-infection in diphtheria, as should also be excluded for the same reason one of the cases of general streptococcus infection, in which the infection-atrium was probably a recent operation-wound in the abdominal wall with abscess formation. Excluding, therefore, these two surgical cases, there still remain 19 cases of poly-infection in which the infection with the bacillus diphtheriæ was complicated by infection with the pyogenic bacteria.

This fact is of importance in connection with the treatment of diphtheria with antitoxin, for while the antitoxin is certainly capable of neutralizing the effects of the diphtheritic poison under certain conditions, yet there is no evidence that it can neutralize the effects of the pyogenic infection as well. If it is generally true that 60 per cent. of all cases die with a general infection with the pyogenic cocci in addition to the infection with the bacillus diphtheriæ, the failure of the antitoxin to effect a cure in a certain proportion of cases can be readily understood. It will be interesting to observe in future, when the antitoxin treatment has come into general use, whether as many cases of pure uncomplicated diphtheria come to autopsy as at present.

Of the 31 cases, broncho-pneumonia was present in 19. The cultures from the lung in these broncho-pneumonias gave the following results:

The bacillus diphtheriæ (not accompanied by other pathogenic bacteria) in 8 cases; the bacillus diphtheriæ and the streptococcus in 5 cases; the bacillus diphtheriæ, the streptococcus and the staphylococcus pyogenes aureus in 2 cases; the bacillus diphtheriæ, the streptococcus, the staphylococcus pyogenes aureus, and the micrococcus lanceolatus in 1 case; the bacillus diphtheriæ and the staphylococcus

pyogenes aureus in 1 case; the streptococcus (not accompanied by other pathogenic bacteria) in 1 case; and the bacillus diphtheriæ, the streptococcus, and the micrococcus lanceolatus in 1 case.

From these results it is apparent that no one organism is to be regarded as the cause of the broncho-pneumonia of diphtheria. In fact, some or all of these species of bacteria above mentioned may be present in the lungs without the occurrence of broncho-pneumonia, as the following results show. Of the 12 cases in which no pneumonic condition was present, the cultures from the lungs in 6 cases (including 2 cases of pulmonary infarction) showed the presence of the bacillus diphtheriæ as the only pathogenic organism present, in 2 cases the bacillus diphtheriæ and the streptococcus, in 2 cases the bacillus diphtheriæ and the staphylococcus pyogenes aureus, and in 2 other cases the bacillus diphtheriæ, the streptococcus, the staphylococcus pyogenes aureus, and the micrococcus lanceolatus.

Undoubtedly a large portion of the bacteria developing in the cultures from the lung are derived from the smaller bronchi, and not from the lung substance proper.

An interesting case, not included in our cases of diphtheria above analyzed, in which the bacillus diphtheriæ was found, may be referred to here. The case was essentially one of broncho-pneumonia in an old man, no other well-marked lesions being made out at the autopsy, although the larynx and trachea were not examined. There were no symptoms during life. The cultures showed the presence of typical forms of the bacillus diphtheriæ in the lung and liver, in addition to various unknown bacteria. The organism, however, both from the lung and liver, was found to have no virulence towards guinea-pigs. This lack of virulence towards guinea-pigs has been occasionally observed in the bacillus diphtheriæ. We have noted it ourselves in a number of instances, even when the bacillus was derived from fatal cases of diphtheria. In addition to this case we have also met with the bacillus diphtheria in the lung in 2 other cases, in 1 of which it was found to be typhically virulent towards a guinea-pig. In neither of these cases was there any suspicion of diphtheria during life, and the air-passages were not examined at the autopsy.

SCARLET FEVER.

Crooke, in 1885, found micrococci in the spleen and kidneys of 30 cases of scarlet fever; and in the same year Fränkel and Frendenberg found the streptococcus in cultures or sections from the submaxillary glands, the liver, the spleen, and kidneys in 3 cases of the disease. Raskin observed the streptococcus in the purulent lymph-glands and joints, and 4 times in the blood of the heart in scarlet fever; and Leubartz examined a case with similar results. In the kidneys, in scarlatinal nephritis, Babes obtained constantly the same organism. The occurrence of a streptococcus general infection has also been constantly observed by Kurth at autopsies on scarlet fever.

Autopsies have been performed on 11 cases of scarlet fever, in 5 of which diphtheria was coincident. A well-marked general infection with the streptococcus was demonstrated in 4 cases (diphtheria being coincident in 2); a general infection with the micrococcus lanceolatus in 1 case; and in another case a general poly-infection with the micrococcus lanceolatus, and the staphylococcus pyogenes aureus; these bacteria being found in the various organs either separately or together. In still another case, in which diphtheria was coincident, the culture from the spleen showed the presence of the micrococcus lanceolatus, and that from the kidney, both the streptococcus and the staphylococcus pyogenes aureus.

In the 4 remaining cases the cultures from the various organs (exclusive of the lungs) were sterile, or contained various unknown bacteria; and no general pyogenic infection could be demonstrated. It may be mentioned that diphtheria was coincident in but 3 of the 7 cases of general infection with pyogenic cocci. Broncho-pneumonia was observed in 9 of the 11 cases. The cultures from the lung in these cases resulted as follows:

The streptococcus was found in 4 cases (including 2 cases of coincident diphtheria in which it was associated with the bacillus diphtheriæ); the bacillus diphtheriæ in 2 cases; the micrococcus lanceolatus in 1 case; the micrococcus lanceolatus and staphylococcus pyogenes aureus in 1 case, and in 1

case a mixture of 3 of these species of bacteria. It is apparent, therefore, that no one species of bacteria is to be found constantly in the broncho-pneumonia of scarlet fever. This has also been shown to be true of the broncho-pneumonia of diphtheria.

TYPHOID FEVER.

But 9 cases of typhoid fever have come to autopsy. The chief points of interest in our study of these cases may be briefly given as follows: The typhoid bacillus has been found in the spleen in 7 of the 9 cases. In the 2 negative cases the failure to find the bacillus may be explained by the fact that both cases had entered the stage of healing. This early disappearance of the typhoid bacillus is interesting. It is worthy of note that in one of these cases there was an acute endocarditis due to the micrococcus lanceolatus. In two cases there was a poly-infection with both the typhoid bacillus and the streptococcus, diphtheria being coincident in one of them. As to the distribution of the typhoid bacillus through the organs other than the spleen, our observations are not as complete as could be desired. Our results on this point may be summarized as follows: The typhoid bacillus has been found in the liver in 4 out of 7 cases in which cultures were made from that organ; in the kidney in 3 out of 7 cases; in the blood of the heart in 2 out of 4; in the mesenteric gland in 3 out of five cases; and in the bile in 2 out of 3 cases.

In the identification of the typhoid bacillus and its differentiation from the bacillus coli communis, great care was exercised, the liability of confusing it with that organism being always borne in mind. The typhoid bacillus in these cases was differentiated from the bacillus coli communis by the absence of gas production in its sugar-agar culture, by the appearance of its potato and litmus-milk cultures, by the absence of indol production, by its motility, and by the ease with which its locomotor organs could be stained. Most of these tests were gone through with in every case to make certain the identity of the bacillus.

BRONCHO-PNEUMONIA AND FOCAL-PNEUMONIA.

The most important and extensive investigations of the bacteriology of the various forms of acute focal inflammations of the lungs are those of Finkler, Netter, and Mosny. Finkler, from an examination of 37 cases of broncho-pneumonia and other forms of acute focal-pneumonia, found that various species of bacteria occurred in these processes, but that the streptococcus was most frequently met with in the focal forms other than true broncho-pneumonia.

Netter studied 95 cases of broncho-pneumonia, 53 of which were in adults and 42 in children. Of the 53 cases in adults, 35 were mono-infections, in which the micrococcus lanceolatus was found in 15, the streptococcus in 12, the bacillus of Friedlander in 9, and the pyogenic staphylococci in 3; while 14 were poly-infections with 2 or more of these species of bacteria. Of the 42 cases in children, 25 were mono-infections, in which the micrococcus lanceolatus was found in 10, the streptococcus in 8, pyogenic staphylococci in 5, and the bacillus of Friedlander in 2; while 17 were poly-infections with 2 or more of these species of bacteria. Netter concludes from his study, that neither in children nor adults is there any relation between the species of bacteria and the type of inflammation in the lung.

Mosny studied 17 cases and found the streptococcus in 11, in 5 cases accompanied by other bacteria; the micrococcus lanceolatus in 4, and the bacillus of Friedlander in 1 case. Three of Mosny's cases were pseudo-lobar, and in these the micrococcus lanceolatus was present.

In the course of this work cultures have been made from the lung in 16 cases in which broncho-pneumonia was present, exclusive of cases of diphtheria and scarlet fever. In 8 of these cases the micrococcus lanceolatus was found as the only pathogenic organism present, in 2 the streptococcus, in 3 the staphylococcus pyogenes aureus, in 1 the micrococcus lanceolatus and the streptococcus; and in 2 only the bacillus coli communis or various unknown bacteria grew in the cultures.

In 2 cases of atypical pneumonic consolidation the micro-

coccus lanceolatus and the streptococcus were respectively present.

From these results it is clear that, as in the bronchopneumonia of diphtheria and scarlet fever, the species of bacteria associated with pneumonic conditions other than the lobar type are subject to great variation.

Of other pathological conditions in the lung, from which cultures have been made, tuberculosis only will be considered. In all of 5 cases of various types of pulmonary tuberculosis either the streptococcus or the staphylococcus pyogenes aureus, or both, have been found in the diseased lung. From 1 of these cases the bacillus diphtheriæ was also isolated, and found to be characteristically virulent for a guinea-pig. No examination was made of the throat or trachea at the autopsy, for there was no suspicion of diphtheritic infection.

XXIII.

SEVEN CASES OF PRIMARY CARCINOMA OF THE LIVER.

By W. H. Prescott, M.D.

On account of the rarity of primary carcinoma of the liver the following cases which have come to autopsy in the last two years seem worthy of mention. Some of them have been found among the autopsies made at the City Hospital, others come from private autopsies. They have been investigated in the pathological laboratory of the City Hospital and in the Sears Laboratory of the Harvard Medical School, under the direction of Dr. Councilman. Cases in which the origin of the tumor was not in the liver itself, but in the gall-bladder, have been included in these cases.

Case 1. — Primary adeno-carcinoma of the liver. I am indebted to Dr. G. M. Garland for the clinical history and for the autopsy of this case.

A. S., 71 years old, musician. Mother died of carcinoma of breast, and one uncle of carcinoma of stomach. A. S. has always enjoyed excellent health, has drunk beer, but not to excess, and for the past two years has drunk but little.

During the summer of 1892 his health began to fail. On July 16th he became partially unconscious upon the street and was brought home dizzy and very much confused in mind; was all right the next day, but weak. There were no cardiac murmurs audible, no albuminuria at the time.

On Oct. 17, 1892, he came to my office on account of indigestion. Examination then revealed a medium-sized ascites which increased rather rapidly in amount. Patient failed steadily in strength until he was obliged to keep his room, which was about the second week of November. At this time his legs and scrotum became quite œdematous and he had difficulty in moving about his room. On November 9 he

was tapped, and one and one-half pailfuls of light straw-colored fluid were removed. Palpation of the abdomen revealed an indurated liver with uneven edge and surface, and also a very hard tumor about the size of a hen's egg on the liver, situated just below and to the right of the ensiform cartilage. The tumor was not tender to pressure. A diagnosis of cirrhosis and primary cancer of the liver was made at this time.

The breathing of the patient was somewhat relieved after the tapping, but he complained of soreness across his abdomen and great thirst. The ascites re-formed with great rapidity, so that the abdomen appeared equally full again at the end of 48 hours. On November 14 he was tapped again in consultation with Dr. S. T. Harmon, and the presence of the hepatic tumor was confirmed. During the night of November 14 the patient was awakened by blood in his mouth, and he vomited considerably more during the night. On November 15 he had several discharges of dark, bloody stools, which were checked by small doses of morphine, and did not occur again. An examination of his chest revealed a small area of dulness in the lower part of the right sub-scapular space, coarse rales and coarse friction rubs were heard over same small area. Nothing abnormal was detected elsewhere in the lungs or heart.

Patient gradually sank into an unconscious condition on November 17, and died Nov. 18, 1892. The whole clinical history of the cancer, therefore, covers a period of only 32 days, but he had himself noticed signs of distention of his abdomen for about two weeks before he first came to me.

Autopsy 15 hours after death. The body very much emaciated. Muscles pale. In peritoneal cavity two gallons of slightly yellowish serum. Liver and spleen free from adhesion.

The heart of normal size; its tissues flabby. On the surface of the endocardium there are numerous small hemorrhages. All the valves of the heart normal save the aortic, in which there are a few calcified areas, evidently due to an extension of the atheromatous degeneration of the aorta to the valves. The aorta atheromatous to a high degree. The coronary arteries thickened, dilated, and thrombosed, especially the descending branch of the left. In the right ventricle there is an adherent grayish-red thrombus.

Lungs free from adhesions. The mucous membrane of bronchi in-

jected, and in the lower posterior portions of lung they contained mucopurulent fluid. In a branch of the pulmonary artery leading to the lower portion of the right lung there is a firmly adherent saddle-shaped thrombus just at the bifurcation of the artery, a leg of the thrombus extending into each division of the bifurcation.

The liver is slightly enlarged. The capsule in places has small masses of connective tissue projecting from it, and is generally thickened and opaque. The surface of the liver is irregular, due to the projection of larger and smaller round or slightly irregular lobular masses. On the anterior surface of the middle line just above the lower border there is a projecting whitish mass of almost cartilaginous consistency. This projects from the liver a distance of 6 cm. and is about the same diameter. On section this mass has a grayish-white color with here and there slightly brownish tints. It is not sharply circumscribed, but projects irregularly into the surrounding liver tissue, so that it is almost impossible to say where the tumor formation ceases. There is a marked formation of connective tissue in the vicinity of the tumor, and it seems to extend into the liver along the masses of connective tissue which pass off from it. Everywhere in the liver the connective tissue is increased, and there are smaller and larger masses of yellow hepatic tissue surrounded by firm lines of connective tissue. The areas of hepatic tissue included by the connective tissue bands vary in size from one to several millimetres in diameter. In most cases this connective tissue is in the form of large bands, and in places smaller bands are given off from these, apparently extending into the lobule and between the cells. The blood-vessels in the tumor and in the surrounding liver tissue are nearly all occluded with firm, slightly adherent masses of thrombi. On following out one of these large veins it is found to be one of the hepatic veins and is entirely filled with the thrombus. This thrombus is firmly adherent over a considerable extent of the surface of the vein, and extends along the vein up to the vena cava, almost filling up the hepatic vein at its entrance into the cava into which it slightly projects. Some of the thrombi can also be followed into the portal vein, almost all the branches of the portal vein going to the right lobe of the liver being thrombosed. The thrombi are generally rather firm, and have a peculiar red, or reddish-white, appearance. In various places in, and in the vicinity of, the tumor spoken of, there are hemorrhages in the tissue. No lesions were found in the hepatic artery.

The gall-bladder was compressed by the large growth previously mentioned; its mucous membrane smooth. The larger bile ducts normal.

The spleen slightly enlarged, of a dark-red color and firm. The kidneys of ordinary size, the cortex slightly contracted. Adrenal glands and pancreas normal.

The stomach filled with large masses of fluid blood. In the mucous membrane near the pylorus there is a small superficial erosion. Fluid blood in the upper part of the small intestine, and in the entire intestinal tract the mucous membrane is stained with blood and covered with mucus. None of the lymph glands in the peritoneum are enlarged.

ANATOMICAL DIAGNOSIS.

Atheromatous degeneration of aorta and of coronary arteries. Calcification of aortic valves. Thrombus in right ventricle. Bronchitis. Carcinoma and cirrhosis of liver. Thrombosis of portal and hepatic veins. Chronic congestion of spleen. Erosion of mucous membrane of stomach. Hemorrhage of stomach.

MICROSCOPIC EXAMINATION.

Sections of the middle of the tumor mass show the chief part of the tissue to be composed of dense masses of connective tissue enclosing epithelial structures. These have in places the appearance of the so-called newly formed bile-ducts in the cirrhotic liver. They have the general structure of tubular glands, the cells being arranged around a lumen, which is frequently filled with masses of detritus. No definite communications are found between these epithelial tubules. In a few places the cells of the tubules are definitely cylindrical, but more generally they have a cuboidal shape, and are of about the size of, or in some cases larger than, the liver cells. In other places this adenomatous structure is lost, and there are large, irregular aggregations of epithelial elements with very small masses of connective tissue dividing them up into groups of a few cells each. Here and there in the connective tissue are very small groups of cells consisting of not more than two or three, surrounded by a firm connective tissue. In many places a transition can be seen between the adenomatous type of the tumor and that of the carcinoma. In these places the epithelial tubules become gradually filled up with cells and converted into solid masses. From these larger masses processes are given off which extend laterally into the surrounding tissue. Only in one or two places in the tumor is there any unaltered liver tissue remaining in the form of definite lobules with a typical arrangement of liver cells. At the periphery of the tumor masses, the liver cells in many places are apparently becoming transformed into cells of the tumor. The cells become larger, their nuclei stain more deeply, and in many two or more nuclei may be seen. The cells of the tumor in general

have a marked similarity to those of the liver, their protoplasm having the same granular appearance. The newly formed cells in the periphery of the tumor lobules seem in most cases to have a distinct granular arrangement which is not so apparent in the older cells in the centre.

In general in the younger portions of the tumor the adenomatous formation is best marked. In some of the tumor masses there is a gradual advancing transformation of the liver tissue into that of the tumor taking place all around the periphery of the lobule. In other cases the tumor formation is more marked in certain places, and in these papillary projections of the tumor extend far into the lobule. In the periphery of the larger tumor the transformation of the tissue of the liver into that of the tumor is very evident, and in places small masses of the tumor are seen at some distance from the main growth. There is everywhere a tendency of the tumor to grow into the blood-vessels. Where the tumor is formed in the periphery of the lobules in many cases it grows directly into the capillary vessels between the rows of cells. Almost all the large vessels, both those of the hepatic and of the portal vein, are filled with tumor masses. These tumor masses are accompanied with thrombosis, and in some places there is a beginning organization of the thrombus. In some of these thrombi the tumor formation is only found at the periphery, and here in many cases a gradual growth into the surrounding liver tissue can be traced. The tumor appears to have first grown into and along the large vessels, and from here to have invaded the surrounding tissue. In all places in and in the vicinity of the large tumor masses there is a marked degeneration of the liver tissue, consisting of fatty degeneration and coagulation necrosis. In many places a considerable amount of hemorrhage is found. There is a marked invasion of leucocytes. In some places this infiltration with leucocytes is so marked that the tissue has rather the appearance of an abscess formation. There are few definite bile-ducts in the vicinity of the tumor. These are lined with cylindrical epithelium which is very much smaller than the epithelium lining the tubules of the tumor, and no transitions can be seen between these bile-ducts and the tumor itself. In the liver, at a distance from the tumor,

and throughout the left lobe there is a well marked and rather coarse cirrhosis. Here there is no special development of the bile-ducts. The so-called newly formed bile-ducts so often found greatly developed in cirrhosis were in this case but little evident.

An examination of the thrombus found in the pulmonary artery shows it to be identical with the thrombi in the veins of the liver. The thrombus represents a tumor mass associated with fibrin formation and other elements of the thrombus. The tumor cells in the thrombus are generally arranged in the form of an adenoma, though in places the carcinomatous form is also evident. There is no extension of the tumor from the thrombus into the surrounding lung tissue. The thrombus found in the heart has the character of an ordinary thrombus.

Case 2. — Primary carcinoma of liver.

Autopsy at City Hospital, January 23, 1895. Case from the service of Dr. George B. Shattuck.

H. A. W., 50, w. Father died of phthisis, mother of cancer of stomach; one brother is living and is in poor health; one brother died of phthisis; one sister is in good health. Patient never had any of children's diseases. Menopause at 45. Appetite always good. Bowels regular. Short of breath for years.

Three weeks ago took cold, began to lose strength; kept up about the house; had a diarrhœa. About one week ago legs and feet became badly swollen. Shortness of breath more marked. Bad pain in cardiac area, but has worked around to the right side; unable to draw a long breath. No cough.

Appetite poor.

Physical examination, very obese. Tongue slightly coated, white. Pulse, fair strength and volume.

Heart. — Dulness over right border of sternum to five and one-half inches from the middle of the sternum. Systolic murmur at apex and transmitted to axilla; systolic murmur over pulmonic area; systolic murmur over aortic area, thence over vessels of neck and along the subclavian. Systolic murmur heard over the sternum at junction with fourth rib.

Lungs. — Moist râles at both bases. Percussion good.

Liver. — Dulness from fifth rib to 1 cm. above costal border.

Spleen. — Eighth rib to costal border.

Abdomen. — Moderate dulness on percussion above symphysis pubis; tympanitic elsewhere, not tender. Considerable œdema of feet. Patella and plantar reflexes present, but sluggish, especially on left side.

AUTOPSY.

Body large, very fat. At the right inguinal region at the junction of the thigh with body, tissue is very hyperæmic, and on the surface is a large bleb filled with clear contents. This hyperæmia extends laterally along the lower portion of abdomen and posteriorly. Legs and feet œdematous. Subcutaneous fat abundant; body walls very thick, especially over the abdomen. Over lower portion of abdomen there are numerous pregnancy cicatrices. Belly walls just above pubis extending slightly to left side and on the right side extending some distance up the flanks are infiltrated with pus.

The fat is softened and filled with pus. This approaches almost to the skin just beneath the vesicle at junction of thigh. It extends apparently along the connective tissue between the fat masses down into the deeper tissue. Only at the place mentioned does it approach the surface. The infiltration extends down into pelvis involving all the cellular tissue here. Peritoneum intensely congested and covered everywhere with slight fibrinous exudation. The exudation mostly over cæcum. Slight amount of fluid in cavity. Diaphragm high. Both lungs adherent by old adhesions. In posterior portion of each, œdema with slight bronchitis.

Heart. — Slightly enlarged. 360 grms. firmly contracted. Myocardium easily torn; valves normal; tissue very yellow and opaque.

Liver. — 1,550 grms. connected to surrounding tissue by old and fresh adhesions; on posterior surface some fibrinous exudation. Gall-bladder filled with thick visid bile. Bile-duct free. Surface of liver everywhere covered with depressions with stellate cicatricial masses extending from these. Everywhere throughout the liver nodular, sometimes circumscribed, tumor masses of grayish color; rather consistent. They are most marked in the right lobe, where they form a large connected mass. The single nodular masses vary in size from a pin's head to the size of an English walnut, and are scattered in every direction throughout the organ.

The lymph glands in porta-hepatis and throughout abdomen are not enlarged. The portal vein somewhat dilated. Veins over surface of the liver and in the suspensory ligaments are also slightly dilated.

Spleen. — Enlarged 370 grms., dark, soft, and almost confluent. Malpighian bodies not enlarged.

Kidneys about normal size; capsule not adherent. On removing the capsule the surface is covered with small granules and deep depressions,

due to an old process. These depressions scattered generally over the surface and do not correspond to infarctions. The surface of the kidney and the section of whitish-yellow opaque color, the markings of cortex obscure, the pyramids pale.

Adrenal glands normal.

Pancreas normal.

Mesentery normal. Veins intensely congested.

Stomach. — Mucous membrane and small intestines generally congested. In lower end of the ileum a few foci of intense congestion with foci of necrosis on surface of mucous membrane. Lower portion of ileum has small areas of necrosis on surface of projecting folds.

Bladder. — Contains a small amount of purulent fluid. The mucous membrane especially around the ureters granular. Right ureter and pelvis of right kidney contains a purulent material. Pelvis slightly dilated.

Uterus is large, the cervical canal is obliterated at the entry into vagina. Cervix dilated forming a cyst filled with muco-purulent material.

Rectum normal.

Pharynx, larynx, and trachea normal.

Cultures from all the organs show general streptococcus infection.

ANATOMICAL DIAGNOSIS.

Erysipelas of thigh with diffuse purulent cellulitis. Streptococcus septicæmia. Fatty degeneration of heart and kidney. Primary carcinoma of liver. Portal congestions. Necrosis of mucous membrane of cæcum. Peritonitis. Œdema and bronchitis of lungs. Acute splenic tumor. Cystitis, pyelo-nephritis on right side. Chronic diffuse nephritis. Anasarca.

MICROSCOPIC EXAMINATION.

The tissues were hardened in various media. Only the results of the fresh examination are given here. Sections of a part of the tumors were cut with the freezing microtome, and examined both unstained and after staining with methyline blue. These sections gave admirable results, and were sufficient to show the nature of the tumor.

On this microscopic examination it was seen that the tumor appeared both in the form of sharply circumscribed nodules and as a more diffuse infiltration. It was more diffuse in the neighborhood of the very smallest nodules than elsewhere. On staining with methyline blue there is a sharp

contrast given between the tumor and the liver tissue. All of the tissue of the tumor taking a slightly lilac color, while the tissue of the liver stains a characteristic blue. The interior of the larger nodules is composed almost entirely of stroma, which stains a distinct, reddish-blue color. In the stroma there are small round or oval spaces, sometimes empty, sometimes filled with cells. It is distinctly fibrous and contains rather few spindle cells. It is much more abundant in the interior of the nodule than in the periphery. In the interior the cells of the tumor are generally degenerated. Their nuclei stain brightly, they are shrunken, and the cells contain larger and smaller fat drops. There is a considerable difference between the tumor in the centre and at the periphery. The cells in the centre of the tumor do not have a characteristic epithelial appearance. Those at periphery have a definite epithelial character and are somewhat smaller than the liver cells. They all contain much fat in the form of small drops, and their nuclei stain more intensely than those of the liver cells, preserving the vesicular epithelial type of nucleus. These cells are arranged in long tubules which frequently communicate with one another, and in their arrangement present much similarity to the general arrangement of the beam work of the liver cells. The tubules are closely packed with the small cells. In some places the tubules of the tumor cells are in immediate contact with those of the liver, and a gradual transition of the liver cells into those of the tumor can be made out. Even in the fresh sections nuclear figures were abundant in the tumor cells in the periphery. The stroma of the tumor is less marked in the periphery, and becomes stronger as it advances toward the centre. The cell masses, however, were much more abundant in the periphery.

The blood-vessels of the liver are in places in immediate relation with those of the tumor, and the stroma appears to result from a growth of the tissue around their walls. In places the liver tissue in the vicinity shows a considerable degree of fatty degeneration, and there is a good deal of bile pigment in the liver cells which is entirely absent in those of the tumor. The cells of the periphery of the tumor have a cuboidal shape in some places approaching the cylindrical,

and are closely packed together. In the vicinity of the larger tumor there are many minute microscopical nodules which are entirely composed of a structure identical with that of the periphery. On all sides the tumor is continuous with the beams of liver cells, and apparently proceeds directly from these. The smaller the masses of the tumor, the more close is the relation of the cells of the tumor to those of the liver. In the periphery of the nodules very large tumor cells are occasionally found, but the relation of these to the tumor could not be made out.

Case 3. — Autopsy for Dr. J. L. Hildreth.

Little is known of the history of this case. For six months before death there were occasional attacks of pain in the abdomen, more marked in the region of the liver, with more or less diarrhœa. There was no ascites during this time. Dr. Hildreth saw the patient three times during the six months. Physical examination showed considerable enlargement of the liver, with nodular masses along the lower border. At no time was there jaundice or any symptoms referable to impaired function of the liver.

Autopsy 24 hours after death. The only lesions found were in the liver. This was very large, weighing 3,400 grms. Everywhere over the surface, particularly over right lobe, there were projecting nodular masses of a grayish-white color. On section many of these masses were sharply separated from the surrounding liver tissue; in other places they blended more or less with this, and the borders of the tumors could not be made out. They varied in size from a pin's head up to a walnut and more. The liver tissue itself was pale, evidently very fat. The markings of the lobules obscure. The gall-bladder was normal, containing a small amount of viscid bile. The cystic duct and all of the larger bile-ducts showed no change.

ANATOMICAL DIAGNOSIS.

Adeno-carcinoma of liver proceeding from bile-ducts. Fatty degeneration and necrosis of liver cells.

On microscopic examination the larger tumor masses were not so sharply circumscribed as appeared on the macroscopic examination, but projected irregularly into the liver tissue on all sides. The liver is intensely fatty. In many places the cells appear as large vacuoles and the nuclei are shrunken. Within the tumor itself occasionally a few scattered liver

cells and rows of them may be recognized by the large vacuoles filled with fat. In the vicinity of these larger tumor masses and elsewhere in the liver are small masses consisting of single alveoli of tumor and groups of alveoli. In the portal spaces there are larger masses which appear to be developed in the portal tissue, and the small masses of the tumor are sometimes connected with these. These smaller tumor masses are more commonly found in the periphery than in the centre of the tumor nodule. In structure the tumor corresponds to a cylindrical cell adeno-carcinoma. It is composed of cylindrical cells generally arranged in a glandular form with a lumen. This more adenomatous structure is found best marked in the smallest nodules and in the periphery of the larger. In other places the alveoli are solid and filled up with cells of cuboidal and irregular shape. In many cases the lumen of the glandular-like structure is filled with a more or less homogeneous mass containing leucocytes, nuclear fragments, and cell detritus. Between these there is a variable amount of fibriller connective tissue with few spindle cells. Occasionally in the midst of the liver tissue there are single cells or groups of two or three cells of the same character as those of the tumor. The position of these cells cannot be made out with certainty owing to the enormous fatty degeneration of the liver cells which obscures the capillaries. In places there is an obvious relation between the tumor development and the periphery of the lobules. The situation of the small tumor masses here corresponds to the situation of the bile-ducts, and no normal bile-ducts can be made out. In addition to the fatty degeneration of the liver cells there is necrosis and hyaline masses, evidently formed by the fusion of several liver cells. The centre of some of the larger masses of tumor is almost taken up by the extensive necrosis which forms a caseous mass composed of necrotic tumor tissue, leucocytes, and connective tissue. In the larger portal spaces masses of the tumor cells are found in what are evidently dilated portal lymphatic vessels. In some of the larger bile-ducts there is a slight tendency to hyperplasia of the epithelium, and there are slight papillary projections into the lumen. In places, there appears also a tendency

to an extension of these epithelial growths in the other direction into the liver tissue.

From the examination of the specimens it seems most probable that the growth should be regarded as having its origin in the bile-ducts instead of the liver cells. Although in general so diffuse, there seems to be no relation between the cells of the tumor and the liver cells. Where the cells of the tumor are directly in contact with the liver cells no changes in these can be made out. The relation of the tumor to the bile-ducts is much more obvious. It seems probable that it arises from the bile-ducts, and it extends in the liver both by successive involvement of this and by a growth along the portal lymphatics, and in some cases also by the blood-vessels.

Case 4. — Clinical history.

S. J., female, aged 27. Entered hospital January 22, 1894. Strong, well-built woman. Always well until last confinement. After birth of child, pain in back and abdomen, lost flesh rapidly, became anæmic. Three days before entry to hospital went to bed on account of severe pain in abdomen. Tumor in right side. Operation showed a tubo-ovarian abscess. Death from general peritonitis.

At the autopsy there was a general peritonitis with fibrino-purulent exudation everywhere. Acute fibrinous pleurisy on right side, congestion of lungs with slight œdema. No lesions other than degenerative found in other organs.

ANATOMICAL DIAGNOSIS.

Operation wound in abdominal wall. Tubo-ovarian abscess with rupture. General peritonitis; fibrous pleurisy; congestion and œdema of lungs. Distention of intestine. Acute parenchymatous degeneration. Adeno-carcinoma of liver.

No tumor formation was found in the liver at the autopsy. In the course of the demonstration of the organ in the further incisions which were made in it, a small number of tumor masses, varying in size from a pin's head to that of a filbert, were found in different places throughout the tissue.

Numerous sections of the liver tissue show only four or five of such masses, which are found exclusively in the right lobe.

Microscopic examination shows that the liver, apart from the presence of the tumors, has little or no change beyond that of a slight fatty infiltration, with here and there a slight increase of connective tissue about the portal spaces. In the immediate vicinity of the tumors the fatty infiltration of the liver cells is more marked, and a few of them appear necrotic. The tumor masses are composed of cells of a more or less cylindrical character, sometimes with a distinct lumen, sometimes forming solid masses. In places these cells are arranged in long rows which variously turn and twist. There are numerous connections between these long rows of cells, and the arrangement is somewhat similar to that of the beam work of liver cells. The cells are considerably larger than the epithelium of the bile-ducts. The smaller masses of the tumor appear to be more in relation with the periphery of the lobules than with the centre. No definite transition between the liver cells and those of the tumor can be made out. There appears in most cases to be a sharp separation between the liver cells and those of the tumor. Nor can a transition between the bile-ducts and the tumor be distinguished.

In this case from the general character of the growth it seems probable that the tumor is derived from the bile-ducts, but this cannot be so certainly made out as in Case 3.

Case 5. — Tumor from liver of child aged five months.

I am indebted to Dr. R. W. Lovett for the clinical history of this case, and for the autopsy.

The patient (P. S.), five months old, was seen on June 3, 1893, in consultation with Dr. F. C. Murphy, of Roxbury, having been brought by him to the Out-Patient Department of the Children's Hospital. The child had been born of healthy parents, but the labor had been difficult, and instruments had been used. The family history was negative and the grandmother had died of paralysis. The child had been well as a baby, and had nursed. Had had no previous illness. About a month before the baby was seen symptoms of indigestion had been present with distention of abdomen and a certain amount of diarrhoea. It was hardly enough to

attract attention until ten days before the child was seen the mother noticed a lump in the upper part of the abdomen, which was sufficient to attract her attention when dressing the child. The child had at that time lost considerable flesh and was looking poorly. The symptoms of indigestion became worse, and five days before the child was seen Dr. Murphy was called in and found what appeared to be the symptoms of intestinal obstruction. There were no movements from the bowels; the abdomen was distended. There was manifest discomfort, and in the course of a day or two vomiting began. At the time the child was seen the abdomen was much distended: the general appearance of the child was bad, as it was white and emaciated. Vomiting had been more or less constant for the past three days, and was becoming worse. The pulse was pretty good, and not of a character to indicate as much intestinal obstruction as the other symptoms would have suggested. At the upper part of the abdomen could be felt a rounded tumor which could not be definitely located, but seemed to fill the whole upper part of the abdomen. The distention of the abdomen rendered recognition difficult.

The child was operated on the following day by Dr. Lovett. The incision was made in the median line, and an enormous liver immediately presented itself. The child's condition was so bad that no exploration of the abdomen was made. A small piece of the liver was removed for examination and the abdomen sewed up. The child rallied well from the shock of the operation, but having been in bad condition at the beginning it died in about twenty-four hours.

The autopsy was made by Dr. Mallory.

No lesions were found apart from the liver. The liver is slightly enlarged. The entire left lobe is occupied by a large tumor mass, and scarcely any liver tissue is visible at this point. The surface of the growth is in general smooth with projecting connective tissue adhesions. The stomach is intimately adherent to the tumor over its entire anterior border. At one point there is an ulceration 5 cm. in diameter where the tumor has grown through the wall of the stomach and projects into its cavity. Its projecting surface is ragged, and in the vicinity there are erosions and irregularities of the mucous surface. The consistency of the tumor mass is rather soft, and on section shows two sorts of tissue; a grayish stroma extending everywhere with irregular masses of a soft

consistency and of an opaque, whitish color imbedded in this. Only in a few places within the tumor are small masses of tissue which may be recognized as liver tissue. The tumor in its greatest diameter measures 8 cm. There is but the one single mass in the tissue. The right lobe of the liver is slightly enlarged, otherwise of normal appearance. There is no affection of either the gall-bladder or the bile-ducts.

ANATOMICAL DIAGNOSIS.

Congenital carcinoma of liver with perforation into stomach.

The microscopic examination of the tumor shows a most interesting and complex structure. The liver at a distance from the growth shows no change. In the immediate vicinity the liver cells are flattened and spindle-shaped from pressure. Throughout the tumor are large masses of fibrous tissue, and in this structures somewhat resembling the bile-duct growth found in cirrhosis can be distinguished. In the periphery of the tumor this connective tissue and apparent growth of the bile-ducts is much more evident. In addition to this fibrous tissue two other sorts of tissue can be distinguished in the tumor. There are large masses of cells of a distinctly epithelial type which are imbedded in the fibrous tissue. These cells are arranged in large masses with a small amount of connective tissue between them. The nuclei of the cells are large and the protoplasm rather hyaline, and they have a general cuboidal and irregular shape. In addition to these cells, and frequently running between them as a stroma, there are groups and bands of cells of spindle shape. The tissue here has all of the characters of a sarcoma. These masses of spindle cells in many places have a definite connection with the fibrous tissue and apparently proceed from this. There are also isolated masses of distinctly hyaline tissue in various places throughout the tumor. This tissue has the appearance of amyloid, but does not give this reaction. It is perfectly homogeneous and hyaline, stains diffusely with eosin and with safranin, and has imbedded in the hyaline masses stellate cells which lie in small cavities, which frequently communicate with one another.

From the microscopic examination of the tumor it seems probable that it began to grow at a comparatively early

period of fœtal life. The major portion of the growth is made up of distinctly epithelial structures probably starting in the epithelium before the differentiation of the epithelium into hepatic tissue had taken place. In addition to this there is a probable growth of the connective tissue resulting in the formation of the sarcomatous-like tissue.

The tumor is further interesting from its relations with the stomach. Cases of perforation into the liver of carcinoma of the stomach and extension of carcinoma of the stomach into the liver by continuity are not very uncommon, but the reverse of this where a tumor of the liver grows directly into the stomach is very rare.

Case 6. — Primary carcinoma of gall-bladder. Autopsy made for Dr. J. L. Hildreth.

Woman aged 43. Mother of four healthy children, and had in general enjoyed good health. Thirteen months before death the patient noticed a small projection, slightly painful on pressure, on the right side just below margin of ribs. She was examined. Diagnosis of probable tumor of gall-bladder was made. Five months later a consultation was held and the advisability of an operation considered. On account of the general condition of the patient and the evidently advanced character of the growth an operation was not deemed advisable. The patient's strength gradually declined, and two days before death there was considerable pain in precardial region with palpitation of heart. There was no ascites, no jaundice or other symptoms of interference with the hepatic function. There was little or no pain at any time.

Autopsy 24 hours after death.

The body somewhat emaciated. Lungs normal. Along the free edge of the mitral valve there are slight reddish projections. In the spleen numerous fresh infarctions. In the kidney there are a few cysts and slight passive congestion, otherwise normal. Transverse colon is firmly adherent to liver and abdominal wall near the hepatic flexure. The mucous membrane of large intestine smooth, the follicles slightly enlarged. At the place of the adhesion of the hepatic flexure smooth, nodular masses can be felt through the mucous membrane, which in most places is freely movable over them. The common duct pervious and somewhat dilated. The cystic duct dilated; its mucous membrane smooth. In the cystic duct there are several larger and smaller

fasceted reddish-brown gall-stones. At the lower end of the gall-bladder the mucous membrane is occupied by an irregular rather firm tumor mass. Imbedded in this tumor mass, which is entirely on the side toward the liver, there is a large gall-stone 4 by 3 cm. The upper portion of the gall-bladder is converted into a thin walled cyst with numerous trabeculæ projecting into the liver, all of which contain small gall-stones. Between the trabeculæ there are large tumor masses continuous with the tumor in the lower end of the gall-bladder. Alongside of the gall-bladder, between the adherent transverse colon and the liver, there is a cavity which contains a thick, gelatinous pus, and numerous small gall-stones. On section of the colon at its adhesions with the gall-bladder there is an infiltration of the muscular coats of the intestine with the tumor. The mucous surface is intact. Almost the entire right lobe of the liver is occupied by a large tumor mass which extends into it from the gall-bladder. In addition to this large mass there are larger and smaller tumor masses varying from 1 to 10 cm. which are scattered throughout the tissue, both in the depths of the tissue and projecting above the peritoneal surface. Many of these tumors entirely surround branches of the hepatic vein, but in no place is there an invasion of the lumen. The abdominal wall is adherent over the gall-bladder and tumor on the right side, but there does not appear to be any infiltration of the abdominal wall with the tumor. The liver elsewhere shows only evidence of chronic passive congestion.

ANATOMICAL DIAGNOSIS.

Carcinoma of gall-bladder with extension into liver and surrounding tissue. Adhesion of transverse colon to gall-bladder with slight stricture. Cholelithiasis. Perforation of gall-bladder with formation of abscess between colon and liver. Acute endocarditis. Infarction of spleen.

Microscopic examination of the tissue shows the structure of most of the tumor to be that of a typical carcinoma, in many respects resembling scirrhus carcinoma of the breast, the alveoli being comparatively small, and the cells rather large. The cells have the general character of epithelium with large vesicular nuclei. The connective tissue in the vicinity of the tumors is so abundant that it gives the appearance of cirrhosis. The nodules are generally sharply circumscribed. In one specimen just at the periphery of the growth there is a bile-duct with both a papillary new growth of the epithelium, and an extension outward into the liver tissue. Many small masses of tumor are found in the portal spaces, showing an obvious connection with the bile-ducts; in others the tumor cells appear to lie in the lymphatics.

In the smallest mass of the tumor and in places in the periphery of the larger masses the cells have a typical adenomatous arrangement with a definite lumen.

Examination of tumor cells with high power shows great variation in size and form. Large cells with numerous nuclei are frequent, and some of these resemble the large cells of the bone marrow. Where the adenomatous character of the growth is best marked, many of the tubes contain a colloid or mucoid material. The nuclear figures are exceedingly common in all parts of the tumor in the specimens hardened in alcohol. Cell division of the tumor cells apparently takes place in a variety of ways. There are not only the perfectly typical figures, but in the large cells with a number of nuclei the most complex figures, evidently indicating a multiple division of the nuclei, are seen. In most of these dividing cells the spindles and centrosomes can be made out with great clearness. No participation of the liver cells in the tumor cell formation can be made out. In the vicinity of the larger nodules the tumor appears to grow by pushing its way into the liver along the capillaries. The liver cells are compressed by the tumor, filled with bile pigment, and undergo atrophy. The examination of sections made between the adherent colon and the tumor show that all of the structures of the bowel are involved up to the mucous membrane, but there is no extension of the tumor into this.

Case 7. — Autopsy for Dr. M. V. Pierce, of Milton.

Woman aged 39. Perfectly well up to seven weeks before death. No history of pain, no jaundice, and nothing referable to any symptoms on the part of the liver. On November 30 there was an attack of pain and vomiting. Shortly before this there had been obstinate constipation. From this time the patient's strength gradually declined. The constipation and the vomiting persisted, but at no time did it have a stercoraceous character. Examination of abdomen at all times was negative. Patient's condition gradually grew worse. Thirty-six hours before death there was more intense pain, followed by collapse, in which condition she died.

Autopsy, January 17. The body well developed, well nourished. Abdomen swollen and tympanitic. The heart and lungs normal. Stomach, duodenum, and transverse colon adherent to the liver. At the point of adhesion in the colon, which is just at the hepatic flexure, the bowel is constricted, its calibre at this point reduced to 5 cm. There is no stricture in the duodenum or stomach. Below the stricture the ascending colon is enormously dilated; the circumference of the cæcum is 17 cm. The peritoneal surface of the intestines covered with a thick fibrino-purulent exudation. This is most marked in the vicinity of the cæcum. At this point the intestines are matted together, and a small cavity formed between the adhesions which contains about 50 cc. of foul pus. The vermiform appendix is 6 cc. in length and greatly enlarged. It is of a dark gangrenous appearance over an area of 3 cm. in diameter, about 3 cm. from the end. In the middle of this area there are two perforations, each about 1½ mm. in diameter. Upon opening the appendix the entire wall is soft and gangrenous about the perforations. No hard masses of fæces or foreign bodies are found. At the point of the stricture of the colon at the adhesions to the gall-bladder there is a depression of the mucous membrane with a dense, firm base, but no ulceration. The hepatic and common duct are pervious in their entire length, the hepatic duct being much dilated. The cystic duct is occluded in almost its whole extent. It can be traced for but a short distance from the common duct, when it becomes enclosed in a mass of dense cicatricial tissue. The gall-bladder is filled with a solid mass of stones varying greatly in size. The two largest of these are round and somewhat separated from the others. One of these was impacted in the cystic duct and the other in a diverticulum, extending laterally from the gall-bladder. These gall-stones were enclosed in a whitish mucoid mass. The wall of the gall-bladder in its entire extent is greatly thickened, white, and dense, and on its mucous surface there are numerous papillary projections. The dense tissue of the wall of the gall-bladder extends for a variable distance into the liver around it. The liver tissue surrounding the gall-bladder is atrophied and cicatricial, and in the cicatricial tissue there are small tumor masses, similar to those in the wall of the gall-bladder. This tissue extended through the muscular coat and up to the mucous membrane of the colon. The lymph glands in the porta-hepatis are enlarged, indurated, and contain infiltrated tumor masses. The tissue of the liver elsewhere is anæmic and rather fatty.

ANATOMICAL DIAGNOSIS.

Peritonitis from perforation. Stricture of large intestine. Gangrenous appendicitis with perforation. Impaction of gall-stones with obliteration of cystic duct. Carcinoma of gall-bladder with metastases in liver and lymph glands. Colon bacilli in the cultures.

Microscopic examination of sections made at various

places in the wall of the gall-bladder and at the cicatrices in the colon show that the tumor is essentially an adeno-carcinoma. In places there is a distinct papillary tumor formation in the gall-bladder. In the most recent portions of the tumor the structure is more that of an adenoma with cylindrical cells. In the course of its development this structure is gradually lost, the cells become more irregular in character, and the typical carcinoma is produced.

Examination of the large intestine shows that the tumor formation has in no place extended to the mucous membrane of this, although it has invaded all the other coats. In the vicinity of the tumor masses around the gall-bladder there is considerable increase in the interstitial tissue. Elsewhere the liver shows a perfectly normal appearance.

The liver may be regarded as one of the organs in the body comparatively free from primary development of carcinoma. In this it follows the general rule that those organs which are the most likely to be the seat of carcinoma metastases have a certain immunity from primary tumors. We can easily see from the circulation of the liver why it should be so frequently the seat of the metastases. Carcinoma of the intestinal canal is comparatively common, and from the relations of the portal circulation to the liver we can easily understand the great frequency of secondary tumor formations in this. Receiving as it does another supply of blood from the hepatic artery, it has in this way another, though less probable, chance for tumor cells to be carried into it.

The seven cases reported here represent the most common and the rarer forms of primary carcinoma of the liver. In three of the cases, the first, second, and fifth, the origin of the tumors is undoubtedly to be referred to the liver cells themselves; and even in Cases 3 and 4 the possibility of this cannot be excluded, although from a microscopical examination it seems more probable that they have arisen from the bile-ducts. Although the epithelium of the liver is highly differentiated, its power of regeneration is considerable, and hyperplasia of the liver cells takes place under a variety of conditions. Ponfick has shown that the greater portion of the liver of a rabbit can be removed and regeneration take place from the remaining portions.

In cases of cirrhosis and other conditions in which there is a destruction of liver tissue, there is considerable hyperplastic growth. There may be new formation of liver tissue occurring in a nodular form in which the structure of the liver may be almost perfectly reproduced. These nodular hyperplasias are not uncommonly seen in connection with cirrhosis, and it is doubtful whether they should be considered as belonging to the true tumors, but rather as representing attempts at regeneration of the liver tissue.

Cases of tubular adenoma have been described by a number of authors. In all of these cases there have been more or less circumscribed tumors in the liver composed of tubular glands, generally with anastomoses between them. The cells lining these glands are generally of a cylindrical character, but this shape of the cells is not so evident in the tumors originating in the liver cells as in those in which the growth proceeds from the bile-ducts. In Cases 1 and 2 it seemed perfectly evident that the growth did take place from the liver cells. The evidence of this is shown in the absence of any changes of the bile-ducts which would point to the development of the tumors from these. In both cases the masses of the tumor cells were in direct contact with the liver cells, and the transitions of these into the cells of the tumor could be made out. In the fifth case the tumor also apparently had its origin in the liver cells and probably started at a very early period of development, when the differentiation of the epithelium into liver cells and bile-ducts had not taken place. There was little or no similarity in the structure of the tumor to the hepatic tissue. The stroma in these cases appears to be formed from a small amount of tissue around the blood-vessels.

A further interesting point in Case 5 is the character of the stroma. In places it resembles so completely the structure of the spindle cell sarcoma, that had such places alone been seen such a diagnosis would have been made. In the third and fourth case the tumor most probably developed from the bile-duct. This is shown from the character of the tumor being that of a cylindrical cell adeno-carcinoma, from the absence of changes in the liver cells, and from the character of its growth. In one of the cases the proliferative

changes in the bile-ducts, consisting of papillary new growths of the epithelium and carcinoma formation, could be made out.

The sixth and seventh case represent by far the most common form of primary carcinoma of the liver. These cases are usually considered apart from the other primary tumors. A great number of such cases have been reported with careful analysis of the clinical symptoms which they cause. An interesting point here is the connection between such tumors and the gall-stones. In both of the cases reported by me gall-stones were present; and in the careful analyses of cases which have been made by Musser and by Siegert, it is shown that gall-stones are present in more than ninety per cent. of these cases. There are two views held with regard to these. Klebs, Zenker, and other authors think that the gall-stones are primary, and that the tumors result from the irritation produced by these. . There is first the adenoma of the mucous membrane with papillary growths in the gall-bladder, and the carcinoma follows these. Other authors assume that the connection is the reverse, and that the tumor in the gall-bladder favors the formation of the stones. There is no doubt that favorable conditions for the formation of concretions may be given by the presence of the tumor. The increased mucoid secretion, and the separation of small necrotic tumor masses from the growth, may give a foundation on which the deposit may take place.

All my cases were seen in women, and statistics show that they are more prone to carcinoma of the gall-bladder than are men. Statistics collected by various authors show that women are more liable in the proportion of 5 to 1. This has been attributed to various causes, the principal one being the irritation of the gall-bladder which is produced by lacing. This it is hardly fair to assume, because certainly in the majority of cases the line of atrophy due to lacing comes above the gall-bladder.

A careful analysis of the symptoms resulting from primary carcinoma of the gall-bladder is given by Ames in an article in the Johns Hopkins Bulletin, June–July, 1894. All of the symptoms produced are usually referable to conditions arising from the presence of the tumors rather than from the

tumor itself. Jaundice is frequently present, and is to be accounted for by the occlusion of the bile-ducts by the masses of tumor. In none of my cases was there any jaundice. In the same way there may be ascites, when the portal veins are compressed by the tumor. This was present in a marked degree in the first case. There may also be gastric disturbances, which may be accounted for by the pressure of the growing tumor masses on the stomach or duodenum. Pain is not infrequently present, but is most marked in those cases in which there is more or less involvement of the peritoneum consisting in a peri-hepatitis with adhesions. In other cases the presence of a tumor mass gives the only indication. The symptoms in general are more marked in those tumors originating in the gall-bladder than those coming from the liver itself.

In the seventh case death was due to stricture of the colon produced by the adhesions. Many of these cases are accompanied by a cirrhosis, and the symptoms may be due more to this than to the tumor itself. In the first case the symptoms were clearly those of cirrhosis, the diagnosis of tumor being made only from the evidence of the tumor on palpation.

XXIV.

TWO CASES OF SYPHILIS OF THE CENTRAL NERVOUS SYSTEM.

By Edward Wyllys Taylor, M.D.

From the Sears Pathological Laboratory, Harvard Medical School.

Although recognized before his time we owe to Virchow our present comparatively definite knowledge of the syphilitic affections of the nervous system. Proof of gummatous growth in the periosteum, secondary changes in bone, the recognition of disease in the meninges, and of tumor formation in them and in the brain itself, Virchow was the first to demonstrate. Later, through the researches of others, the part played by the blood-vessels attracted its share of study. Differences of opinion prevailed as to the origin of the pathological process, whether from the endothelium (Heubner) or from the connective tissue of the adventitia (Köster, Baumgarten). These questions remain in a measure undetermined; their further discussion does not at present concern us.

Before entering upon a description of the two cases under consideration it will be well to enumerate briefly the chief forms in which syphilis of the nervous system occurs, first in the brain, including disease of the bone and meninges, and secondly in the spinal cord, with an account of our knowledge of its pathological histology.

a. Disease of the bone (skull) occurs primarily as periostitis, formation of gummatous tumors, and necrosis. This is much less usual than secondary ingrowths from the periosteum. Symptoms on the part of the nervous system from such affections of the bone are less common than formerly supposed.

b. More usual are syphilitic new growths proceeding

from the meninges, or originating in the brain (*vide* Case 2). The internal surface of the dura mater is ordinarily affected. The two layers of the dura may be separated by the new growth (*vide* Case 2). The pia is secondarily involved, as a rule, also the cortex and white matter of the cerebral hemispheres.

c. Rarer are tumors of the brain substance itself, unassociated with connective tissue, except such as occurs in the walls of the vessels.

d. Diffuse syphilitic meningitis of the base is of much more frequent occurrence than that of the convexity.

e. Syphilis of the spinal cord manifests itself primarily as a chronic meningitis. The process usually starts from the pia, more rarely from the inner side of the dura. It is characterized, as elsewhere, by the formation of granulation tissue which tends to a thickening of the membranes, with gluing together of nerve roots, and adherence of membranes and spinal cord. The thickening of the membranes is irregular in distribution. Secondary infiltration of the cord leads to characteristic areas of local, peripheral degeneration, with the usual secondary system degenerations if injury to the nerve elements be sufficiently great. In the course of the process gummata may be formed.

f. Primary gumma of the cord is rare.

g. Of great importance is the frequent localization of the syphilitic process in the large blood-vessels, thickening of their walls, their partial or complete occlusion, with the consequent destructive lesions of the parts supplied by them. The much-disputed point as to the origin and character of the process in the walls of the vessels cannot find discussion here. It represents a prolific field for further research.

The pathological histology of syphilis of the nervous system has received an exhaustive study at the hands of Rumpf. ("Die Syphilitische Erkrankungen des Nervensystems," Wiesbaden, 1877.) His conception in brief is as follows:

The analogy of the syphilitic process to that of tuberculosis is, in certain respects, so close that we are justified in assuming the presence of an irritative agent (bacterial?) which produces the original focus of inflammation. The process is best observed about the smaller vessels and capillaries.

In its first stages is seen, often while the lumen is still normal, a collection of cells (leucocytes, round cells, epitheloid cells) about the vessels in the perivascular spaces. The cells increase rapidly in number, and a granulation tissue with delicate stroma is formed, which extends into the adjoining tissue. Some of the cells take on the character of connective tissue cells, until finally a granulomatous tumor is formed. The effect of this rapid growth is to lead to pressure with partial or complete obliteration of the original vessel. With the consequent cutting off of blood supply the new growth begins to undergo its retrograde metamorphosis, softening, cheesy degeneration, necrosis of cells, with ultimate outcome in caseation, or more often in the formation of firm fibrous tissue, poor in cells. (For examples of these various processes *vide* Case 2.) All of this may occur in much the same way in tuberculosis. Distinguishing features are the much greater tendency in tuberculosis to purulent degeneration, and to the formation of small circumscribed tumors (miliary tubercles). Such are the general characteristics of the syphilitic new growth as seen in the nervous system, with variations dependent alone upon the character of the tissue (brain, meninges, or cord) in which the process occurs."

To summarize the pathological anatomy as given by Rumpf:

1. Syphilitic disease of the nervous system has its origin always in the vessels. The new-formed tissue belongs in the category of the granulomatous tumors. Syphilitic infiltration of the connective tissue capillaries and of the vessel capillaries is to be distinguished.

2. At times the process leads to the formation of more or less circumscribed tumors, which at a certain stage of retrograde metamorphosis are known as gummata. Oftener to more or less extensive infiltration without definite tumor formation.

3. The existence and the retrograde metamorphosis of the syphilitic infiltration is dependent upon the amount of blood supply to the affected part.

4. Inflammation of connective tissue and of vessel capillaries necessarily leads to different results in its effect on the general nervous system.

Case 1. — Service of Dr. F. S. Watson, etc.

E. McK., aged 34. Entered hospital March 15, 1894.

Present illness of five days' duration, began with a swelling under left ear, from which the redness and swelling extended from day to day over the face. There had been more or less incontinence of urine for several months.

On physical examination the patient was well developed and nourished. Temperature on admission 104, pulse 160, feeble and irregular. Left ear swollen and red. From this, swelling and redness extended over entire surface of face. Eyes are swollen and is not able to open them. Heart's action weak and irregular. No murmur. Urine highly colored. Specific gravity 1024. Increased amount of urea. Slight trace of albumen. No sugar, a few hyaline and granular casts. Patient gradually became weaker in spite of all treatment. Temperature continued high, pulse 120, feeble. Death in five days.

The autopsy showed well-marked erysipelas extending over face, scalp, neck, and upper part of thorax. All of the organs amyloid, very extensive visceral syphilis. The scalp unusually adherent; skull thick. Over parietal bone on left side there is a hard elevated round nodule 1½ cm. in diameter and projecting ½ cm. over surface. The dura smooth, the pia slightly cloudy and not adherent. Pacchionian granulations well marked. The convolutions of the brain normal. The cord throughout firmer than normal. In the lower portion of the cord extending into the cauda equina there is considerable formation of connective tissue binding the nerves together. Newly formed tissue is firm and œdematous.

ANATOMICAL DIAGNOSIS.

Erysipelas of scalp and face. Cicatrix on penis.
Erysipelas of lung (gumma and syphilitic pneumonia).
Syphilis of liver (gummata and diffuse).
Syphilis of kidney (gummata and diffuse).
Syphilis of adrenals (diffuse).
Syphilis of testicles (gummata and diffuse).
Syphilis of seminal vesicles (gummata and diffuse.)
Atrophy of vas deferens.
Chronic contraction of bladder with cystitis.
Slight pyelonephritis.
Syphilis of lumbar cord and nerves of cauda equinea.
Syphilitic periostitis of tibia and skull with new bone formation.

Cord given for microscopic examination. Hardened in Müller's fluid and alcohol. Cut at various heights from conus terminalis to upper cervical region. Stained with ammonio-carmine; picric acid-fuchsin (Van Giesen); hæmatoxylin (Weigert).

MACROSCOPIC APPEARANCE ON SECTION.

Deformity, especially of posterior aspect of whole sacral cord, possibly artefact. Destructive degeneration of both gray and white matter. Nerves of cauda equina apparently matted together.

MICROSCOPIC EXAMINATION.

(*Conus Terminalis — Weigert Preparation.*)

Cord much deformed posteriorly and laterally. Anterior fissure widened by the interposition of a dense, wavy, fibrous tissue, which represents the greatly thickened pia. This tissue surrounds the cord, and in places extends into its substance. It contains numerous large, essentially normal, gaping vessels. In addition to these are many smaller vessels with much thickened walls, often difficult to distinguish from the surrounding connective tissue. Such vessels — capillaries — are also to be found in large numbers in the cord itself, especially in the parts which have undergone the greatest connective tissue change. White matter as well as gray at this level almost completely destroyed, its place being taken by neuroglia and thick, old connective tissue. About the periphery, especially anteriorly, are a few medullated nerve fibres, most of them in varying stages of degeneration. Central canal occluded. Nerves of the cauda equina which lie nearest the cord, viz., those given off lowest down, are somewhat degenerated. Nerve bundles are separated by connective tissue, which reaches its greatest thickness on the posterior aspect of the cauda equina. Vessels of the cauda equina for the most part normal.

Picric acid-fuchsin preparation, at slightly higher level shows general thickening of the pia with a great amount of connective tissue growth into the substance of the cord. This ingrowth proceeds especially from the posterior side. Septa of connective tissue (stained red) permeate the cord in every direction, giving it a curious mottled appearance. Numerous blood-vessels. Connective tissue, though much less marked, extends about the periphery of the cauda equina and between certain of the nerve bundles. An occasional vessel has enormously thickened walls, staining homogeneously (hyaline, amyloid). Bundles of nerve fibres in the neighborhood of the cord show numerous degenerated areas. Deformity of the cord persists.

Hæmatoxylin preparation shows the degenerated areas to be made up of dense connective tissue and neuroglia, whose formation has evidently been compensatory, to take the place of destroyed nerve tissue. In those parts where the connective tissue is densest are numerous vessels of small calibre, containing a few red corpuscles, having greatly thickened walls, and surrounded by connective tissue in such a way as to make their identification difficult, and their outlines often impossible to distinguish. There is no definite cell infiltration about the vessels and no tumor (gumma) formation. The nuclei are many of them oval and spindle shaped. The whole process shows evidence of being distinctly chronic in character.

Lower Sacral Cord. — Cord still much deformed and even more completely degenerated than in lower section. Occasional multipolar ganglion cells visible, whose nuclei and nucleoli are only rarely to be made out. An area of tissue in the gray matter of one side has broken down and fallen out in cutting. The connective tissue formation is almost completely confined to the posterior aspect of the cord. Thick-

ened vessels of small calibre are everywhere to be seen. At certain places the nerves of the cauda equina are intimately connected with the cord by means of connective tissue; fibrous hyperplasia of the pia.

Mid and Upper Sacral. — The intensity of the process begins to diminish. The pial thickening is less marked; the extension into the cord is not so great as at a lower level. The anterior horn of one side is practically normal (Weigert prep.), with numerous well-formed and nucleated ganglion cells. The whole posterior part of the cord and the other anterior horn are destroyed. The connective tissue ingrowth is no longer prominent, the degenerated areas in all probability being made up of a great increase in neuroglia, giving a mesh-work appearance (carmine-hæmatoxylin) with numerous small and round nuclei (neuroglia cells). Central canal pervious. Cord flattened on one (the more degenerated) side and drawn out posteriorly.

Lower Lumbar. — Shows a tendency toward return to the normal. One anterior horn and surrounding white matter of normal appearance. Degenerated areas are traversed by dense bands of tissue (neuroglia processes) which run antero-posteriorly. Perivascular lymph spaces widely dilated. Pia posteriorly markedly thickened, otherwise normal. Cord slightly flattened laterally.

Lumbar Enlargement. — One-half of the cord practically normal except few strips of degenerated, tissue in posterior median column. Opposite side flattened. Space in gray matter between anterior and posterior horn much degenerated, with degeneration of ganglion cells in that part. Degeneration in posterior-median, lateral, and anterior white columns. Fibrous thickening of pia posteriorly. Vessels normal. Large central canal. From this point upwards the cord becomes rapidly normal. In upper part of lumbar enlargement there is slight degeneration of posterior median column of both sides, probably secondary. Pia normal. Cord of normal shape.

Throughout the dorsal region there is extremely slight degeneration along either side of the posterior fissure. The pyramidal tracts, more marked on one side, are stained slightly lighter with Weigert than other white matter. Slight secondary degeneration of unknown cause.

Longitudinal sections of the cauda equina show marked degeneration of many nerve bundles.

To summarize: We have to do with a localized destructive lesion of the sacral and part of the lumbar cord occurring in a person showing widespread visceral syphilis. The process in the cord is characterized by alterations in blood-vessels; by the formation of a large amount of connective tissue; by thickening of the pia, and by degenerations of essential nerve tissue without and within the cord. Apart from insignificant degenerations in the white matter, the remainder of the central nervous system, so far as examined, is normal.

Etiologically there is nothing of importance except the statement that the patient is said to have had "incontinence of urine" following a "fall" ten years ago. All details are lacking. The possibility of a blow on the back giving rise to the pathological process under consideration should, however, be considered. In the absence of any definite data, and in view of the fact that no sign of fracture or dislocation was found at the autopsy, entirely apart from the evidence of the lesion itself, we are justified in ignoring this possibility.

A question of greater importance is the relation between trauma and the outbreak of latent syphilis. Such association has been observed in a number of well-studied cases, and may be of significance in the present case. Here again speculation is useless, owing to the uncertainty of the clinical facts.

In all probability we have to do with a syphilitic cord lesion of unusual location, associated with profound syphilis of other organs. The lesion is evidently an old one, and represents the ultimate connective tissue change of an acute syphilitic infiltration probably originating in the pia. This is the common outcome of a syphilitic cord affection of chronic course, and in itself presents nothing remarkable.

The points to which special interest attach are:

1. The unusual location and limitation of the process.
2. The remarkable involvement of the cord, supposing the process to have originated in the pia.
3. The changes in the blood-vessels within the cord.

No special significance is to be attached to the deformity of the cord; it is improbable that it is to be regarded as an artefact, and in any case would have no bearing on the pathological process under consideration.

The incontinence of urine observed during life was doubtless due to a destruction of the bladder reflex in the lower lumbar cord (2–4 lumbar segment, Starr). There is no statement in the brief clinical report as to the condition of other reflexes, nor of sensibility. Apart from the bladder disturbance the history affords no other symptom of neurological interest.

Case 2. — J. F., 38 years.

Admitted for the second time into hospital, December 26, 1894, in the service of Dr. C. F. Folsom.

Clinical diagnosis. Cerebral gumma.

Patient entered hospital for first time November 19, and on December 13 was discharged to the Out-Patient Department with symptoms relieved. Entered hospital again December 26. On first admission, November 19, gave history of small-pox and typhoid fever 20 years ago. Alcoholic habits. Has six living children; has had four still-born children. The children born as follows: First two still-born at full term. Next four living children, then two still-born, and following these two living children. Had a bubo before marriage. Denied venereal disease.

Present illness began four weeks ago. She first noticed that her left arm was tightly squeezing the baby she was holding, and was twisted outwards, causing great pain. Her left leg at the same time quivered. The attack lasted three or four minutes, and was shortly followed by another similar attack. A few days afterwards a third attack similar to the two preceding ones. She felt dazed, but was conscious. After these attacks the arm seemed numb. Could move hand and arm, but they remained weak. Two weeks after this, leg felt weak and the loss of strength in leg gradually became greater. Two days after attack, over vertex and right side of head there were severe and frequent attacks of pains, neuralgic in character.

On physical examination, patient well developed and well nourished. Right pupil larger than left. Both react alike. Tongue moist, with white coating. Pulse regular, good strength and volume. Heart sounds normal. Lungs normal. Patella reflexes active and normal on both sides. Plantar reflex absent on left and diminished on right. Scar in groin. Left arm partially paralyzed. Treatment, iodide of potash grains 20 t. d. On November 30, in evening, patient had several slight convulsions, but was better in morning. On December 2 much better, and walked length of bed. On December 10 could walk well, could control hand and arm, but still considerable weakness. December 13 discharged to Out-Patient Department. December 26 readmitted. Patient had been gaining strength every day up to fourth day before admission. On this day she had general clonic convulsions lasting over an hour. Has felt fairly well since, but remained in bed. Much headache since convulsions. For two nights has not slept well, owing to headache.

Physical examination. Pupils equal and react to light. Mouth slightly drawn to left side. Tongue to one side. Pulse regular and of fair strength and volume. Heart somewhat enlarged, apex at fifth intercostal space, inside nipple line. Sounds at base blowing in character. Pulmonic sounds accentuated and reduplicated. Abdomen lax, tympanitic resonance increased throughout; more marked in median line. Liver dulness normal. Partial paralysis of left arm and leg. Patella reflexes present and equal. Plantar reflexes present. No ankle clonus.

December 29. Since entry patient has had headache most of time. Right pupil is larger. Tongue turned somewhat to left side. Left arm

showed marked atrophy and sensation is diminished. Patient cannot move herself so well as when previously discharged from hospital. Grasp of left hand very weak.

January 2. Headache and depression of spirits. Appetite poor.

January 6. Had slight spasm.

January 7. Right pupil somewhat larger. Tongue deviates to left side. Left arm more rigid than before. More stupid than at any time since first entry into hospital. Fluid runs from right side of mouth in spite of her efforts to retain it.

January 9. Patient gradually failing. Died in the evening.

Autopsy.— The dura generally adherent especially in the right parietal region. At this point there is an area 5 cm. in diameter where the upper surface of dura is roughened and shows numerous roughened elevations. Inner table of skull corresponding to this very porous and red in color. The entire skull apparently more porous and brittle than normal. On the internal surface of dura on right side in parietal region corresponding to the external thickening there are a number of nodules from the size of a pea to that of a bean adherent to pia and projecting into the adjoining cortex with which they seem to be agglutinated. The nodules are firm and yellowish on section. The cortical surface of the brain at this point is firmer, denser, and slightly yellowish. This infiltration extends inward about 1 cm. over irregular areas in the region of the fissure of Rolando, and near its upper extremity. The internal surface of this area is about 3 cm. from the longitudinal fissure. No other lesions found in the brain.

The remainder of the autopsy showed nothing beyond a slight diffuse nephritis. No amyloid.

ANATOMICAL DIAGNOSIS.

Gummata of cerebral meninges involving brain. Chronic diffuse nephritis.

Syphilis (gumma) of dura mater and cortex cerebri.

Specimens of diseased dura and cortex hardened in alcohol, corrosive sublimate, and Müller's fluid. Stained with hæmotoxylin-eosin. After staining, areas of necrosis are clearly visible in the dura with the naked eye. At thickest point the dura measures 5 mm. Cortex shows two areas of necrosis (gummata) separated slightly and each measuring about 5 mm. in diameter. One of these areas reaches to the surface, the other is situated slightly deeper, with a layer of less infiltrated tissue intervening between it and the surface of the brain.

MICROSCOPIC EXAMINATION.

Dura mater much thickened, as stated above, and irregularly. The new-formed tissue has apparently arisen from the layers of the dura; in several places both on peripheral and visceral aspects it has infiltrated through, involving the bone on one side and the cerebral cortex on the other. The new-formed tissue is densely infiltrated with cells, with nuclei of varying size and shape. This infiltration, except in occasional instances, bears no apparent relation to blood-vessels. Except about

the periphery vessels are not numerous. Occasional vessels of small calibre in the new growth show narrowed lumen and thickened walls. Infiltration about vessels is not so conspicuous as in the cortex, indicating the greater age of the dural growth. The stages of the retrograde metamorphosis are well shown. In places is seen a dense cell infiltration with the formation of a delicate stroma. In others the stroma is more apparent, the cells less numerous and of a more elongated form. In still others the stroma is dense, the nuclei few, with final formation of a dense scar-like connective tissue. In addition to these definite stages of connective tissue formation are other areas in which cells are undergoing necrotic changes with fragmentation of nuclei, the whole staining diffusely with hæmatoxylin. There is no actual caseation. Occasional giant cell.

Cortex.— Evidently the process here is secondary to and immediately associated with that of the dura. The pia is naturally included in the lesion. Especially worthy of note in the cortex is the infiltration about the capillaries, marking the earliest stage of the syphilitic process. This is especially to be seen in those parts of the section which are at some distance from the distinct tumor formation. In most cases the walls of the capillaries are entirely normal. Later stages of the process do not differ in the cortex from what has been already described as taking place in the dura. Ganglion cells with well-defined nuclei and nucleoli are to be seen in the cortex outside the defined tumor tissue.

In this case we have to do with the frequent primary involvement of the dura mater with secondary encroachment on the cerebral cortex leading to the formation of more or less sharply defined gummatous tumors. With the exception of slight adherence of the dura near the base of the skull nothing further was found at the autopsy attributable to syphilis. There was no macroscopic disease of the vessels. The spinal cord was not examined.

The clinical symptoms in this case are well explained by the pathological findings. The patient had suffered for a month or more with spasm of the left arm and leg, accompanied by weakness and some disturbance of sensibility. With this was intense headache, often neuralgic in character, localized on right side of head and vertex. Then gradually loss of power in the leg followed by decided improvement under treatment. Discharged to Out-Patient Department, and readmitted two weeks before death, during which previous symptoms had recurred with much mental depression.

The extensive involvement of the dura doubtless explains the persistent headache; its point of greatest severity cor-

responding to the position of the greatest dural thickening. The initiative symptoms, spasm of left arm and leg, find their explanation in the disease of the dura and pia with consequent pressure over the motor areas of the right hemisphere. The paresis with remissions under treatment, but for the most part progressive in character, was due to the more or less complete destruction of the motor cortex along the Rolandic fissure. It should be borne in mind that the area of impairment of cortical function was undoubtedly greater than that immediately about the definite tumor formation, as indicated by the involvement of both arm and leg.

To conclude: In this case we have to deal with a more or less generalized primary affection of the dura mater over the convexity of the brain, with localized gumma formation both in the dura and brain over the right fissure of Rolando at its upper part. Points of interest are the non-implication of the larger vessels and the definite localizing symptoms during life. Operation, had it been attempted, might have led to definite though probably temporary amelioration of symptoms.

XXV.

INFECTIOUS DISEASES, AND THE MANAGEMENT OF HOSPITALS DEVOTED TO INFECTIOUS DISEASES.

Report of a Committee appointed by the Staff of the Boston City Hospital.

This report is divided into, *first*, a general consideration of contagious diseases which are treated in the Boston City Hospital.

Second, a review of the information obtained from various hospitals and physicians to whom questions relative to contagious diseases and their hospital management were directed.

Third, an article on the clinical history of those cases of angina which resemble diphtheria, from observations on cases in the Boston City Hospital.

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The diseases treated in the contagious department of the Boston City Hospital are diphtheria, scarlet fever, and measles. First it will be well to consider briefly the features of these diseases which have a direct bearing on their contagiousness. This would include a consideration of their pathology, the infectious agents concerned in the production of the lesions, the manner in which these infectious agents cause the lesions of the disease, the character and locality of the lesions, the habitat of the infectious

agents in the body, their duration of life, the possibility of their existence outside of the body, and the various ways in which they may enter into the body and produce the disease. Our knowledge with regard to the infectious agents in these diseases is much more certain and definite in diphtheria than in scarlet fever and measles. While our knowledge of the infectious agent in diphtheria and the pathogenesis of the disease may be regarded now as based on exact knowledge, we know little or nothing of these features in the other two diseases. From what we know of infectious diseases in general, it may be assumed for the present that scarlet fever and measles are also due to specific microörganisms which are in some way given off from the infected individual, and enter into the bodies of other individuals, producing the disease. The conclusions concerning infection, etc., which we are justified in drawing from diphtheria, may be transferred with probably slight modifications to the other diseases.

DIPHTHERIA.

Diphtheria is produced by a bacillus having definite morphological and cultural peculiarities. The proof of this has now become so strong that the fact need not be questioned. The proof is given by the fact that in cases of true diphtheria, the bacillus is always found associated with the typical primary lesions of the disease. The bacillus can be grown in suitable media outside of the body, and when animals are inoculated with pure cultures of the bacillus, a disease in all respects similar to the disease seen in man may be produced.

In many of the infectious diseases we must distinguish between the lesions which are produced in some parts of the body where the disease is localized and caused by the action of pathogenic agents at this point, and secondary lesions which are due to these primary lesions, and which may or may not be associated with the presence of the organisms. This is notably the case with regard to diphtheria. From the first knowledge which we had of the disease the presence of certain lesions on the mucous membrane of the pharynx, or air-passages, was recognized as its essential characteristic.

These may be spoken of as the primary lesions of the disease. This primary lesion appears in the form of a whitish or grayish-white membrane, sometimes slightly elevated, sometimes not, which cannot be removed without leaving a loss of substance. The mucous membrane in the vicinity of this membrane is usually swollen, hyperaemic, and presents other evidences of inflammation.

The membrane consists of a fibrinous exudation mingled with pus cells and necrotic epithelium. It is produced by a fibrinous inflammatory exudation coming in contact with a necrotic mucous membrane. The most frequent situation of this membrane is in the uvula and tonsils. It may extend from here into the larynx and upper air-passages, both or either of which may be the seat of the disease. In addition to these lesions of the mucous membrane there is usually more or less swelling of the lymph glands of the neck, and in some cases an œdematous infiltration of the tissue about them. In cases of death from diphtheria we almost always find a general swelling of the lymph glandular tissue of the body, which is most marked in the solitary follicles and Peyers patches of the intestines. The lungs, after death, are always found implicated to a greater or less extent. In rare cases a definite pseudo-membrane may be found in the smaller bronchi which does not represent a continuous extension from the larynx, but may be found without any involvement of the larynx or trachea. There is always a broncho-pneumonia of greater or less extent and atelectasis dependent upon this. On microscopic examination various lesions consisting in very minute necrotic foci are found in all of the viscera.

In a certain number of cases paralysis of various muscles takes place some time after apparent recovery. The same paralysis, though usually involving other groups of muscles than those affected in man, may be experimentally produced in animals by the injection of the chemical poisons of the bacilli. These paralyses have been found to be due to degenerative lesions in the nerves, of the same character as the necrotic foci found generally in all of the organs.

The diphtheria bacilli are found in the primary lesions of the disease. They are found in great numbers in the pseudo-membrane, and are usually absent in the tissue im-

mediately beneath this. In the earlier investigations made on diphtheria it was supposed that the presence of the bacilli was confined to this membrane. It has since been shown from the work of various observers, the most important of which is that contained in the communication of Dr. Wright on the work done at the City Hospital, that the bacilli are found in other places. Their presence in the body, apart from the local lesions, is more or less accidental, and is by no means constant. Microscopic examination and cultures from the membrane show their presence in enormous numbers. They are contained in such small numbers in the tissues that they can never be made out on microscopic examination. Cultures made from the kidneys and liver, the brain, and other organs, may or may not show the development of a few colonies. In cases of pseudo-membraneous formation in the bronchi, they may be found here in the same abundance as in the pseudo-membrane elsewhere.

From the work of various observers, beginning with that of Roux and Yersin, it has been shown that the internal lesions of the disease, and probably most of the symptoms observed during life, such as fever, cardiac weakness, etc., are due to toxic chemical substances which are produced by the bacilli in the local lesions, and which are absorbed and taken into the circulation. Similar toxic substances are also produced in the cultures of the bacilli outside of the body, and similar lesions may be produced by the inoculation of animals with these soluble chemical substances alone. We know little or nothing concerning the exact character of these toxic substances. It has usually been maintained that they are albuminous in character and in a way related to the organic ferments. In cultures it has been held by some that they are formed by changes in the culture media itself, by others that they are due either to a substance secreted by the bacteria, or they represent a solution of the chemical substances of the bodies of the bacteria themselves after they have died. These toxic substances act in all respects as chemical poisons, and the severity of the lesions produced are directly proportionate to the dose and the virulence of the poison.

Pure, uncomplicated cases of diphtheria, due to the presence of the diphtheria bacillus alone, may be produced by experimental inoculation of animals with pure cultures. They are rarely seen in man. Along with the presence of the diphtheria bacilli, we find always in the fatal cases, and in the majority of the observations made during life, the presence of other organisms which are themselves pathogenic. The most important of these other organisms found associated with diphtheria bacilli are the various forms of streptococci, the staphylococci, and the pneumococci. These may be confined to the membrane, or they may enter into the circulation, producing a definite septicæmia. The constant broncho-pneumonia found in fatal cases is due to some one of these organisms; and they are also found in cultures made from the various organs after death.

In addition to these typical cases of diphtheria there may be other lesions due to an atypical localization of the diphtheria bacilli. Two cases of acute endocarditis have been reported: one in Baltimore by Howard, and the other a case at the City Hospital reported by Wright in which diphtheria bacilli were found in the cardiac lesions. Such cases, however, are so rare that they may be considered in the light of curiosities.

Not only will the diphtheria bacilli produce the lesions spoken of in the mucous membranes, but similar lesions may be produced, and on the exterior of the body, where the bacilli come in contact with a tissue deprived of the protection of the epidermis. We have found in the cases at the City Hospital numerous instances in which small wounds and abrasions on the surface of diphtheria patients have become infected with the bacilli, and similar lesions to those in the pharynx and due to the bacilli have been produced. In these cases small excoriations about the nostrils and lips in diphtheria patients and slight abrasions elsewhere on the body have become infected. Probably the cases which have been reported of diphtheritic endometritis, vaginitis, and conjunctivitis in which the diphtheria bacilli have been found belong to such accidental infections. Such cases have an important bearing on the infectiousness of the disease.

THE LIFE DURATION OF THE BACILLI.

The diphtheria bacilli do not belong to the class of organisms which have the greatest power of resistance. The greatest resistance to influences injurious to bacteria are found in those species in the course of whose life spores, analogous to the seeds of higher plants, are produced. The diphtheria bacilli do not produce spores, and hence they are in general more easily destroyed than such organisms as the anthrax or tetanus bacilli. Their duration of life outside of the body in cultures may in general be assumed, when the cultures are carefully protected from the influence of light and other agencies more or less injurious, to be about eight to ten months. Cases, however, have been reported in which living bacilli have been found on various objects some years after these have become infected, and such objects have proven sources of infection for the disease. Thus a case has been reported in which the playthings used by a child with diphtheria were used by another child ten years afterwards and produced the disease. It is probable that sometimes outside of the body the bacilli may find more suitable conditions for the retention of their vitality than can be given in the artificial culture media, and we are not justified in taking as the limits of their vitality what can be learned of them in the laboratory.

INFECTION IN DIPHTHERIA.

We know that this disease should be regarded as one of the most contagious diseases, and we can easily see from the knowledge we have gained of it why this should be the case. The bacilli are not only contained in the membrane in the throat, but are found in large numbers in the buccal and nasal secretions of the infected individual. From the abundant opportunity which is given for these secretions to come in contact with external objects, the bacilli are easily conveyed to the surroundings of the patient. Even with the greatest care which can be exercised in the management of such patients the secretion containing the bacilli may come in contact with the floor of the ward, the bed-clothing, etc. This may take place in the acts of coughing, expectorating,

and in various other ways. The organisms may certainly be assumed to be constantly upon the hands of the patients from the frequency with which the hands and the fingers come in contact with the nasal and buccal secretions. Evidence is given of this from the not infrequent cases in which various other localities of the body are infected, it being only possible for the infection to take place by the hands. One of the most striking cases of this was shown at the City Hospital by the production of a typical pseudo-membrane in the anal fissure of a diphtheritic patient. Other cases have been seen in which abrasions of the skin produced by scratching have become covered with a true diphtheritic membrane. The bacilli are only contained in the secretions which come from contact with the diseased mucous surface. The urine and feces of the affected individual are assumed to be non-infectious, though we do not know of any exact experiments which have been made to prove this.

Investigations carried out in the diphtheria wards in the City Hospital have shown that from the diphtheria patients the bacilli can infect a considerable area about the patient. They have been found in a broom with which the wards have been swept, and on the shoes, clothing, and hair of the nurses in charge of the patients. The positive proof which has been given in this way of the extension of the bacilli beyond their immediate surroundings would justify us in regarding the source of infection shown as much broader and more general than would at first sight appear. In investigations of this sort the presence of the bacilli can be determined in only a very small area. They have been found on the shoes of three nurses in the diphtheria ward and on various other objects. When we consider how small a portion of such objects can be subjected to investigation, we may be justified in assuming that they are present in every case. For instance, the area which can be definitely subjected to investigation, and on which we can say the diphtheria bacilli are or are not present, would probably be not more than one-half a square centimetre. The importance of this positive proof of the diffusion of the diphtheria bacilli which was first given by the investigations made at the City Hospital, and of the presence of the diphtheria bacilli in such

places, cannot be overestimated. Park has also found the bacilli on the bed linen of diphtheria patients. It has generally been assumed that the virus of diphtheria is not volatile; that infection does not take place from the air, but from actual contact with the patient or intermediate objects which are infected with the bacilli: and when we think of the infinite number of chances which are given for infection in this way, it is not necessary to assume transmission by air. A large series of investigations which have been made at the City Hospital of the dust which accumulates in the ward on flat surfaces, as on mantles, window-ledges, and various other places, have invariably shown the absence of the bacilli, and although it cannot be stated definitely from these negative experiments that the bacilli were not present, it is probable, that if they were present in such places, they were there in extraordinarily small numbers.

The patient himself is, not only during his illness but for a considerable time afterwards, a source of infection. It was first shown by the experiments of Tobiesen that, for a long time after apparent complete recovery, the bacilli were still found in the buccal secretions, in a condition of full virulence. It has also been found to be the case in the City Hospital, where patients are kept after apparent recovery until bacteriological examination has shown that the bacilli have disappeared from the throats. This period in some cases is extended up to eight weeks after apparent convalescence. Where this is not controlled by bacteriological examinations, patients after complete recovery from diphtheria, as far as physical signs and symptoms permit us to judge, should be kept as carefully isolated as during the height of the disease, for at least six weeks. We believe that it should be the function of such an infectious hospital to do the utmost amount of good for the community. Not only should the sick of the community find relief at the hospital, but the community should be, in addition, protected from disease to the greatest extent possible. At the same time, in such a hospital various researches on the infectious diseases, with a view to a better understanding of their nature, their mode of extension and treatment, should be carried out. If we compare the work done by the hospitals in the simple treatment of disease

with this broader work, the first is of minor importance in comparison.

In certain of the infectious diseases it is probable that infection does not usually take place on a perfectly normal mucous membrane; but preceding infection by the specific organism, a condition favoring their action must be produced by some antecedent pathological condition. In cases of infection of the external surface of the body with the diphtheria organisms, it is certain that this must be the case. We know that where the surface is deprived of this protection, the diphtheria bacilli are capable of producing their typical lesions. It has been assumed by some authors, rather than based on experience or experiments, that this may be the case in the infection of the pharynx and air-passages. This is certainly the case in animals. The typical local lesions of the disease will not be produced when the bacilli are placed in contact with the normal mucous surface. The conjunctiva is the only exception to this. Observations have also shown us that, in some, although probably rare cases, the diphtheria bacilli can find entrance into the mouths of individuals, and can exist there, without the production of any local lesions. In observations made by Tangl on the mucous membrane of the mouth in 400 normal individuals, he only found them twice; and other observers have shown that their presence in normal individuals is an extremely rare occurrence. Other observers, particularly Roux and Yersin and Hoffman, have found an organism which they regard as a modified form of the diphtheria bacillus much more frequently. Thus Roux found it 26 times in 59 perfectly normal individuals, in a city in which diphtheria was not prevalent. It is doubtful if these observers had the true bacillus, or even a modified form of this. Their work has not been confirmed by later investigators. The general experience which we have had at the City Hospital is opposed to this. In their presence on normal mucous membranes, diphtheria bacilli show a similarity to the organism of croupous pneumonia which, it is well known, is found in a large number of cases of normal individuals. There can be little doubt but that such cases are capable of spreading the disease.

Investigations have been made at the City Hospital hav-

ing in view the determining of the frequency with which the bacilli are found in the mouths of those in any way connected with the infectious wards of the Boston City Hospital, even though they may show no lesions.

At various times during last October Dr. J. H. Wright made cultures from the noses and throats of nurses, physicians, and internes in the diphtheria ward; that is, ward E, of the Boston City Hospital.

Nineteen cultures were made from the noses, all of which were negative.

Eighteen cultures were made from the throats, and in one case the K.L. bacilli were found. This nurse was not ill, and the examination of the throat showed only a slight reddening of the fauces. The organism was tested on guinea pigs and found to be virulent. At the same time the cultures which had been made previously from various objects in the ward were repeated.

The shoes of twelve nurses were examined, and in one case a virulent K.L. bacillus was found. Cultures made from the hair, from the nose, from the brushes and mops used in the ward, and from the floor of the ward itself, were all negative.

In this connection it might be interesting to refer to some cultures made by Dr. Wright from families in the practice of Dr. Williams. In four such families in which diphtheria had developed, and in which all of the members of the family were exposed to contagion, two of the cultures were negative and in two diphtheria bacilli were found in one or more individuals without the clinical evidence of the disease.

Inoculations made with the organisms so obtained showed them to be of full virulence.

DISINFECTION WITH A VIEW TO THE DESTRUCTION OF BACILLI.

Disinfection experiments have shown that the bacilli are not capable of great resistance to the action of germicides. They may be destroyed by almost any of the ordinary approved methods of disinfection. They are especially affected by the action of sunlight. Exposure of them to the direct rays of the sun, either in cultures or infected articles, de-

stroys them in a few hours. They are also destroyed by weak solutions of corrosive sublimate and by carbolic acid. Infected articles may be disinfected by exposure to superheated steam or to heat. Where this is not available 1-5000 corrosive sublimate or 5 per cent. carbolic acid may be used.

The question of the management of diphtheria is rendered much more complicated by a condition which, though not peculiar to diphtheria, assumes more importance in this disease than in other diseases.

In diseases generally we are able to recognize the character of the disease by certain local manifestations. The anatomical lesions which the pathogenic agent produces are more or less characteristic for every disease, and from these anatomical lesions alone we are able to recognize the disease. This is not the case in diphtheria. The essential and primary lesion of diphtheria, and the only lesion which is open to inspection, is the pseudo-membranous inflammation of the throat. As we have said, this membrane formation is due to a combination of necrosis with fibrinous exudation. The fibrinous exudation comes from the sub-mucous tissue and coagulates on the surface, forming with the necrotic cells a dense, firm membrane. It has long been known that such a membrane may be formed in other conditions. The application of strong caustics to certain of the mucous membranes will produce a condition closely resembling that produced in diphtheria.

Pseudo-membranous inflammation of the throat, the primary anatomical lesion of diphtheria, is also met with in the course of other diseases. It is more or less common in scarlet fever, in measles, and it may also be found in typhoid fever, or it may be found without any accompanying disease. These pseudo-membranous inflammations, not associated with the presence of diphtheria bacilli, have of late attracted a great deal of attention, and have been the subject of repeated investigation. Various bacteria have been found associated with them. A form of micrococcus growing in chains, the so-called streptococcus, appears to be the organism most frequently concerned in their production. In some cases the ordinary micrococcus of suppuration, the

staphylococcus aureus, may be found, and in still other cases the pneumococcus. These cases in general have a rather benign course. The onset of the inflammation is frequently marked clinically by more stormy symptoms than is the case in true diphtheria. The temperature and pulse may be higher, but the cardiac weakness and general depression so often found in diphtheria is usually absent. Where the cases are recovered from, they are never followed by paralysis or any of the sequelæ of diphtheria. The frequency of such cases has been differently estimated by various writers on the subject. It is probable that some have overestimated their frequency and importance. There is nothing in the extent and in the distribution of the pseudo-membrane which can distinguish them from diphtheria. The membrane may be found in the larynx, or it may be found in the pharynx in connection with the affection of the larynx, or alone. The majority of the cases of the so-called membranous croup, in which there is a membrane confined to the larynx, have been found to be due to the diphtheria bacilli. In a certain number, however, the diphtheria are absent, and the streptococci have been found alone. That such cases are common in association with measles and scarlet fever may be due to the fact that in these diseases the general resistance of the mucous membrane of the throat is lowered by the exanthematous eruption and a better opportunity given for the action of various organisms. The cases are probably rather more uncommon in adults than in children.

In a certain number of cases of pseudo-membranous inflammation which have been observed in the City Hospital diphtheria bacilli have not been found in the examinations made in the first few days, though they subsequently appeared. This may be due, on the one hand, to a possible infection which has taken place in the hospital, and on the other to the fact that diphtheria bacilli were present in such small numbers that they were overlooked on first examination.

Not only do we find the anatomical lesions of diphtheria, as shown by the pseudo-membrane, in certain of the infectious diseases, but cases of mixed infection between scarlet fever and diphtheria, measles and diphtheria, and even

typhoid fever and diphtheria have been found among the cases at the City Hospital. In some cases it is possible that infection has taken place in the hospital itself. In other diseases we may have the same anatomical lesions produced by different causes. We may have cases of broncho-pneumonia due to entirely different causes, in which anatomically the lesions may be precisely the same. The same thing is also true in a number of skin diseases; although here the ease with which the pathological lesions can be investigated is very much greater than in the case of a disease located in the throat. A disease is defined and classified, not only by the presence of certain anatomical lesions, but much more surely by the definite ætiological factor. The ætiological classification of disease is much more satisfactory than any other, although it must in the beginning be productive of confusion. There appears to be little doubt in cases of mixed infection that the presence of other organisms can greatly increase the gravity of the prognosis in diphtheria.

Some attempt has been made to trace the relationship between the severity of the disease and the readiness of infection, but no constant relation can be shown between the two. Certainly infection would seem to take place from some cases with a great deal more ease than from others. Dr. Wright has made investigations to determine if there be any relation between the virulence of the bacilli as tested on animals and the severity of the case from which they were derived, by making cultures from a series of cases and inoculating animals with these cultures. He has found that in general there is no relation between the virulence of the bacillus as tested on animals and the severity of the disease.

It has also been found that in not a few instances the diphtheria bacilli may be present in the throat and the typical lesions of the disease may not be produced. Cases are found of inflammation of the pharynx, varying in degree, in which there may be no trace of false membrane, and in which fully virulent diphtheria bacilli are found. These cases are especially dangerous because they are not accompanied by any symptoms of the disease, and what might be clinically and anatomically regarded as a slight attack of angina may prove the source of infection of numerous cases of malignant

diphtheria. This still more complicates the matter, for it shows that not only may various other organisms produce the same lesions as the diphtheria bacilli, but these may be present and cause only an inflammation without any definite character, or in some cases it would appear that no inflammation at all is produced. We have a completely analogous instance given by the pneumococci. These are present in full virulence in the mouths of many normal individuals without producing any lesions, or they may produce the most different pathological conditions.

There is frequently difficulty in making a sure diagnosis of diphtheria. In a number of cases there will be no difficulty at all. If the patient comes to the hospital with a diphtheritic membranous throat and with the clinical condition which may be recognized as due to the absorption of the diphtheritic poison, there is no difficulty in diagnosis, and the patient may be transferred without further delay to the diphtheria ward. In certain cases there is difficulty in diagnosis.

Nearly all of the replies which have been received from physicians, and the general expression of opinion in the recent Congress held at Budapest, were that in many cases a bacteriological examination was necessary to establish a sure diagnosis. It would probably be necessary to make a bacteriological examination to establish the diagnosis of a large number of the cases which are brought into the hospital. It must be recognized in the beginning that our present methods for making the bacteriological diagnosis are far from perfect. The making of such examinations becomes routine work, and it is exceedingly difficult to have routine examinations made with sufficient care. The greatest care is necessary in making the examinations, and the position of the examiner is one of great responsibility, especially when the diagnosis is absolutely based on the bacteriological report. Care must be exercised in obtaining the specimen from the throat. If the interne is careless in his work it is possible that he may not place the swab in contact with the diphtheritic membrane, and hence may fail to obtain a good specimen. Great care must be exercised in making the culture, although this is usually stated in books to be an exceedingly easy procedure;

any one who has had much experience in making these examinations will be convinced that this is not so. A number of cases may be met with which will present no difficulties in the bacteriological diagnosis. Then a series of cases will appear in which it is exceedingly difficult to say whether or not the organisms found are diphtheria bacilli, and in some cases this can only be told by inoculating guinea pigs. If the method is to be of any great value the examination must be carried out in the most thorough and careful manner, otherwise it is better to have no examination at all; but by confining the examination to the cases which really present difficulties in diagnosis it can be thoroughly made. In many cases a single examination is not sufficient to certainly establish the diagnosis. Cases have repeatedly been seen at the City Hospital in which a single examination has failed to demonstrate the presence of the bacilli, while subsequent examinations have shown its presence. It is true that in these cases there has always been the chance that the bacilli were acquired in the hospital, but they have frequently been found so short a time after admission that this would seem to be hardly possible. The amount of the bacteriological work becomes much increased from the examinations of the convalescent patients for bacilli.

In scarlet fever the cause of the disease remains to be discovered. We know that the disease is a typical infectious and contagious disease, and that cases do not arise except from contagion. We are, of course, not nearly so well informed owing to our ignorance of the cause, and the part of the body in which the disease is definitely located, and from which the organisms causing it are given off. There are two distinct lesions of the disease: those located in the throat, and those located in the skin. It has been generally assumed that the virus of the disease may be given off from the skin, and that it may be entangled with the dry epidermic scales which would be carried everywhere by currents of air, and therefore more easily than in diphtheria. This, though generally believed in, is denied by some observers, who hold that the skin eruption in this disease is due to reflex nervous action, and that the cause of the disease should not be looked for here. They attach much more im-

portance to the secretions from the mouth for conveying the disease, than to any material emanation which might be given off from the skin. In any case the virus would seem to be one of considerable tenacity of life. It is known that it can be conveyed on infected clothing. Cases have repeatedly arisen which prove this beyond any reasonable doubt. The infectious substance is able to retain its full strength a considerable time, the length of which is not known so well as in diphtheria.

We should apply all the knowledge which we have gained with reference to diphtheria to this disease. It is certain that infection can take place in the same way, and it is probable that it can also take place by the air. While from what we know of diphtheria it would seem to be probable that we could expose an individual in a ward full of this disease, and have him attended by some persons who come in no contact with the disease, with but little probability of infection, the same is not true of scarlet fever. Whether or no the other secretions of the body, as the urine and feces, can act as carriers of the disease, is not certain.

All that has been said of the contagion in scarlet fever and diphtheria applies with equal force to measles, although in this we do not know the exact cause of the disease, nor its exact location in the body. Still we do know that cases of measles never arise *de novo*, and that in some way a substance capable of producing the disease is given off from the body. It is generally estimated that the area of contagion, so to speak, is greater in measles than in either diphtheria or scarlet fever. In any hospital in which these three diseases are taken there is the greatest danger of mixed infection. One disease may be complicated with another and render the prognosis much more grave. Repeated instances of such mixed infection have arisen at the City Hospital.

W. T. Councilman.

With the view of collecting the experience of other hospitals in the management of contagious diseases, a series of questions were sent to various hospitals in which infectious diseases were taken along with other diseases, and to hos-

pitals in which contagious diseases alone were treated, and to physicians.

The following questions were addressed to the hospitals in which infectious diseases were treated along with other diseases:

1. What disposition is made of the named infectious diseases after their admission to the hospital?

2. Have you a separate ambulance for these cases?

3. Are these cases isolated from the rest of the hospital or from each other?

4. Have you observed cases of mixed infection of scarlet fever and diphtheria, or measles and diphtheria, or scarlet fever and measles, and if so, are such cases isolated?

5. Have you observation rooms in which doubtful cases can be isolated?

6. If the answer to any of the above questions be negative, has it seemed to you that cases of infection have arisen in consequence?

7. Have cases of infection occurred in the general wards apparently to be referred to the infectious wards? If so, how has the infection seemed to have been conveyed?

8. What means are taken to prevent infection? In cases of accidental infection arising in the general wards, are these wards disinfected? How often are the infectious wards disinfected, and what is the method of disinfection? Is personal disinfection of medical officers and attendants in these wards carried out, and is the dress changed when in the wards?

9. Who has the medical care of these wards? Have they separate internes, nurses, and servants, and how are these persons isolated from the rest of the hospital?

10. How long after convalescence are patients kept before discharge? What means are taken for disinfecting patients and their clothing on discharge?

Questions relative to Infection.

1. Is diagnosis in diphtheria based on bacteriological examination?

2. If so, are cases kept in observation rooms pending examination?

3. What has been your experience as to the proportion

of cases which resemble diphtheria anatomically, but in which the K.L. bacillus has not been found?

4. Is the clinical history and prognosis in such cases different from those in which this bacillus is found?

5. Have you reason to believe that such cases are infectious?

Twenty-eight replies were received from hospitals in America, England, Australia, France, Germany, and Italy. Some of the answers in this class come from hospitals in which only contagious diseases are treated and should be considered under that class. A great many of the replies were not signed, so that it was impossible to find from what hospital the replies came.

Answers relative to Hospital Management.

(1.) In regard to the first question the general answer was that when infectious cases are received in the hospital, as soon as they are recognized they are removed either to separate wards or to pavilions separate from the rest of the hospital. In some of the institutions infectious diseases are not generally received into the hospital, but there is an infectious ward or isolated building kept for such cases when they arise within the hospital. In St. Thomas' Hospital, London, which is built on the block system, the end block is reserved for the reception of patients with certain infectious diseases. Diphtheria is admitted into the hospital, but neither measles nor scarlet fever is admitted. Although cases of these two diseases which originate in the hospital are sometimes met with, these are promptly transferred to the infectious block. This portion of the hospital was built expressly for the purpose, and consists of small wards, each of which will contain about a dozen persons. When diphtheria patients are admitted they are sent to a ward which receives such patients only.

(2.) Most of the hospitals have not a separate ambulance for the conveyance of infectious diseases to the hospital. In some cases the patients are brought to the hospital in public conveyances and seemingly in an extremely careless manner. In some cities the hospital does not provide for the convey-

ance of such patients at all, but on notification of the police the cases are sent to the hospital by the health authorities. When ambulances are used, it was generally stated that they were disinfected after each trip, the disinfection being carried out, when the method was stated, by washing with carbolic acid.

(3.) In regard to the third question, referring to the isolation of these cases, in most cases the isolation was effected by separate buildings or by separate wards. The different diseases were isolated from one another in separate wards. In St. Thomas' Hospital all the infectious cases are in a separate block, and in this block there are separate wards for the reception of specific febrile diseases. From the replies of some of the hospitals it is evident that isolation is very imperfectly carried out.

(4.) Most of the answers to this question were positive. In St. Thomas' Hospital it is said that the most frequent combination seen was diphtheria and scarlet fever, diphtheria and measles, the diphtheria as a rule being the primary affection. Such cases were removed from the ward and placed in an empty large ward or in a so-called small ward. These small wards contain two patients and open into a corridor leading to the large ward. They regard a diphtheria patient as peculiarly susceptible to both scarlet fever and measles.

(5.) In St. Thomas' Hospital doubtful cases are placed in the small wards, and when these are occupied the patients are sent to other wards, the character of which is not stated. In the Royal Infirmary, Edinburgh, there are special observation wards separated entirely from the rest of the building, in which doubtful cases appearing in the wards can be isolated until their nature is ascertained.

(6.) The replies to the sixth question were generally indefinite.

(7.) In quite a number of the hospitals the answer to this question was positive. It was supposed that infection was possibly conveyed by allowing physicians to visit both places, or by allowing internes in the contagious ward to visit the general wards. In St. Thomas' Hospital, capacity, 350 beds, about 15 patients a year contract infectious diseases while in the hospital. Diphtheria is stated to be more com-

mon than scarlet fever. On one or two occasions, nurses who were removed from the infectious block to the main building were thought to have conveyed the disease. In this hospital it has never been proved that a medical officer has been the cause of any outbreak in the general wards.

(8.) In most of the hospitals a case of contagious disease accidentally developing in the hospital is removed. The bedding of the patient is always removed with him, and in some cases the ward is closed and disinfected. In St. Thomas' Hospital this is not done when a single sporadic case arises, but is done when several cases follow in succession. The number of times during the year that the wards are disinfected varies, as does also the method. In many cases reliance is placed on disinfection by sulphur, but the most common method used is that of washing the walls and floors with carbolic acid, 5 per cent., or with bichloride solution.

(9.) The replies received to this question vary considerably. In most of the hospitals the contagious cases have separate house officers and nurses who are not allowed to come in contact in any way with the other cases. In some the nurses are separate, but the house officers and visiting physicians are the same. In St. Thomas' Hospital the same physicians and internes have charge of the other wards, and the only precaution used to prevent contagion is that the visit to the contagious wards is made last.

(10.) No definite time is given during which patients are kept after convalescence. Only in a few of the hospitals are the convalescent diphtheria patients controlled by bacteriological investigation. In most cases the time diphtheria patients were kept after convalescence would seem to be too short.

Answers relative to Infection.

(1.) In the majority of these hospitals the diagnosis of diphtheria is controlled by bacteriological examination, and when this is not the case, some of the replies say that they think it should be so. In none of the English hospitals from which replies were received are bacteriological examinations made; they were made in the German, French, and Italian hospitals.

(2.) In some of the hospitals patients were kept in observation rooms pending the bacteriological examination.

(3.) In St. Luke's Hospital, New York, it is stated that since bacteriological examinations have been instituted, about one-half of the cases presenting the anatomical features of diphtheria were found not to have the K.L. bacillus.

To the fourth and fifth questions of this category the answers received were not satisfactory.

The following questions were addressed to the hospitals in which contagious diseases alone are treated:

1. Is the hospital independent, or is it under the supervision of a general hospital?
2. How is the staff of officers made up?
3. Are the medical and executive officers separate?
4. Do the former consist of a visiting staff or of resident physicians? Do they receive compensation, and what is the length of service?
5. What is the proportion of nurses and internes to the patients, and what is their length of service?
6. What is the ambulance service?
7. Have you observed cases of mixed infection of scarlet fever and diphtheria, or measles and diphtheria, or scarlet fever and measles; and if so, are such cases isolated?
8. Have you observation rooms in which doubtful cases can be isolated?
9. Have cases of infection among medical officers, nurses, and servants been observed, and in what proportion to numbers?
10. How often are the wards disinfected, and what are the methods of disinfection used?
11. Is personal disinfection of medical officers and attendants carried out, and is the dress changed when in the wards?
12. How long after convalescence are patients kept before discharge? What means are taken of disinfecting patients and their clothing on discharge? Have you a convalescent home?

Questions relative to Infection.

1. Is diagnosis in diphtheria based on bacteriological examination?

2. If so, are cases kept in observation rooms pending examination?

3. What has been your experience as to the proportion of cases which resemble diphtheria anatomically, but in which the Klebs-Loeffler has not been found?

4. Is the clinical history and prognosis in such cases different from those in which this bacillus is found?

5. Have you reason to believe that such cases are infectious?

Answers relative to Hospital Management.

(1.) In New York there are two hospitals: the Willard Parker Hospital for diphtheria and scarlet fever, and the Riverside Hospital for other contagious diseases. There is also a reception hospital for receiving patients with various diseases, where they are held for observation and afterwards sent to some of the other hospitals. These two hospitals are both under the control of the Board of Health of the city, and are not attached to any general hospital.

(2.) In the Borough Hospital of Nottingham there is one resident medical officer, one assistant matron, 24 nurses, 10 ward maids, 9 men, and 8 servants. The health officer of the Borough is the medical superintendent of the hospital, and has general control. In the most of the English hospitals the health officer of the Borough is the general superintendent of the hospital, and in addition to that there are resident physicians and visiting physicians.

(3.) In none of the hospitals from which we have replies are the executive and medical officers separate.

(4.) In many of the hospitals there are no visiting physicians. In one of the English hospitals the visiting physicians are appointed permanently for seven years and receive 100 pounds yearly. The resident medical officer in the English hospitals receives generally about 250 to 300 pounds and board. In the hospital at Lyons the visiting physicians receive 2,000 francs and their luncheon.

(5.) In the Nottingham Hospital the proportion of nurses is 1 to 5 patients, and when the entire staff is taken it is 1 to 3. In the Willard Parker Hospital, New York, it is about 1 to 10. This is the smallest proportion of nurses in any of the hospitals from which replies were received. In Sheffield it is 1 to 4; in Birmingham 1 to 4 or 6; and in the hospital at Denmark from which we have returns it is 1 to 5. In Lyons it is 1 to 4½ for measles and scarlet fever, and 1 to 6 for diphtheria.

(6.) The ambulance service in some of the hospitals is separate for the separate diseases. In others the ambulances are disinfected after use each time. In some a nurse always goes with the driver. In the Norway Hospital every disease has its separate ambulance. This is possible when the diagnosis is first made and the hospital notified.

(7.) In the Nottingham Hospital there have been but 3 cases of scarlet fever and diphtheria among 1,000 patients with scarlet fever; 1 of measles and diphtheria among 20 of measles. There have been several instances in which scarlet-fever patients have taken measles at intervals from the second to the sixth week after scarlet fever. In general when such mixed infections take place the cases are isolated.

(8.) In most of the hospitals there are rooms for observation of doubtful cases. In the City Hospital of Sheffield there are 12 such rooms. In but 2 instances were there negative answers to this, and in both of these it is stated that changes were under contemplation in which this would take place.

(9.) In the Nottingham Hospital it is stated that one-half of the nurses who had not previously had scarlet fever contracted the disease in the hospital. In the Birmingham Hospital it is also stated that scarlet fever is common among the nurses. In the Leeds Hospital the number of cases of infection among the staff varied from none in 1885 with 223 admissions, to 32 in 1891 with 406 admissions. In the Sheffield Hospital 2 or 3 cases a year have occurred, which have been attributed to carelessness on the part of the internes, they having eaten food, etc., while in the ward. In the Norway Hospital in seven years 3 cases of diphtheria infection have been seen.

(10.) In the Nottingham Hospital a great deal of care is exercised in the disinfection of the wards; the floors are polished, cleaned with 5 per cent. carbolic solution once a month. ("The lower half of the walls is formed of glazed brick, and is cleaned in the same way at the same time. The wards are disinfected by burning sulphur once a year, or oftener if any outbreak of any disease has taken place in a ward. Medical officers keep a suit of clothes for use in the ward and about the hospital premises, and change on leaving the premises. The same rule applies to nurses and others of the staff, who have uniforms when on duty. These must make a complete change before going out. Washing of the hair is not enforced. Macintosh overalls are provided for medical officers, and put on when in the wards. On discharge, clothing of patients is disinfected by heating in steam sterilizer; patients are washed in $\frac{1}{2000}$ corrosive, and patients bathed in bath-room especially for the purpose. We insist on the nurse who does the discharge bathing wearing a special bathing macintosh to prevent infection with contact of dress.") In other hospitals disinfection is carried out completely at least twice a year, and as many times more as is necessary. In the Willard Parker Hospital disinfection is done whenever possible. In the Sheffield Hospital each ward is thoroughly disinfected every year, and the smaller rooms frequently disinfected; sulphur fumigation here is not relied upon. The walls, ceiling, and floor are carefully washed with potash and corrosive. In the Dublin Hospital the wards are disinfected four to six times a year. In the Norway Hospital the observation rooms are always disinfected when a patient is discharged. The method of disinfection in this hospital is by washing with hot water and soft-soap, followed by a solution of carbolic acid

(11.) In Birmingham the clothes used in wards are changed on leaving the hospital. Baths are generally taken before leaving the hospital, but are not insisted upon except in cases of small-pox. In the Leeds Hospital all nurses and servants wear disinfected clothing outside of the hospital, and are expected to bathe before leaving. The medical officers wear overalls and carry out the same rules as for the

nurses. In the City Hospital, Sheffield, personal disinfection is thoroughly carried out, no article of uniform being worn when out of the wards. In all of the hospitals it is stated that personal disinfection is carried out, but the methods are not stated.

(12.) The rule varies; scarlet-fever patients are kept until all desquamation and all discharges have ceased. In the Sheffield Hospital there is a large convalescent home attached to the hospital. In Brighton, scarlet-fever patients are kept until desquamation is completed, which is usually about seven weeks. Other patients, it is said, are kept until free from infection, but the period is not given. In the Willard Parker Hospital, and in the Norway Hospital, patients are kept until free from K.L. In every case the patient is not allowed to leave until thoroughly washed, and then he puts on disinfected clothing.

Answers relative to Infection.

(1.) Bacteriological examination in diphtheria is carried out only in the Willard Parker Hospital and in the Norway Hospital.

(2.) The cases are kept under observation until the diagnosis can be confirmed.

(3.) In these infectious hospitals there seems to have been but few instances in which the K.L. bacillus has not been found in cases resembling diphtheria anatomically.

The fourth question, when answered at all, is answered in the affirmative. The fifth question is answered sometimes in the affirmative, and sometimes in the negative.

The following questions were addressed to physicians:

1. Have you seen cases of mixed infection between scarlet fever and diphtheria, scarlet fever and measles, and diphtheria and measles?

2. In your opinion should diagnosis in diphtheria be based on bacteriological examination?

3. What has been your experience as to the proportion of cases which resemble diphtheria anatomically, but in which the Klebs-Loeffler bacillus has not been found?

4. Are the clinical history and prognosis in such cases different from those in which this bacillus is found?

5. Have you reason to believe that such cases are infectious?

6. Have you reason to believe that in any given case any of these infectious diseases have been transmitted to persons outside of the hospital, the hospital serving as the focus for infection?

To these questions forty-nine replies have been received. Most of them have been from physicians in America. No replies were received from the questions sent to England, to France, and Italy. A number of replies from Germany were received.

Answers from Physicians.

(1.) With regard to the first question the reply was in almost every instance positive. Almost all observers found mixed infection between scarlet fever and diphtheria, and diphtheria and measles. Mixed infection between scarlet fever and measles appears to have been extremely rare, although several observers have seen such cases. Some observers have regarded the mixed infection between diphtheria and measles and scarlet fever as common, but the general opinion is that between scarlet fever and measles it is much more rare. O'Dwyer thinks that measles cannot be treated under the same roof with other diseases, no matter how large the building. It will surely spread. Morse, who has carefully investigated these mixed cases in the Boston City Hospital, said he has known of many mixed cases of scarlet fever and diphtheria in the Boston City Hospital, and thinks that about all of these originated in the hospital. "In a few, however, cultures from swabs taken from the throats of scarlet-fever patients soon after admission showed K.L. The condition surrounding these cases is unknown. I have examined a number of scarlet-fever throats seen in private patients bacteriologically and have never found K.L. Have known of several cases of scarlet fever and measles in the Boston City Hospital, the measles always being secondary." All observers agree that such cases are very much more common in hospitals than in private practice. Dr. Buckingham says he has seen all these cases of mixed infection, but only in hospitals.

(2.) With regard to the second question, most of the replies were affirmative. Adams, of New York, says that the diagnosis should only be made after bacteriological examination. In cultures made in 32 apparently healthy throats the K.L. bacillus were found by the Board of Health in five cases. These cases were from an asylum, and were apparently in perfect health. In 19 apparently healthy throats of children coming to the out-patient department of a New York clinic, the K.L. were found by the Board of Health in two cases. Only one of these cases showed subsequently any clinical symptoms of true diphtheria; yet all were called true diphtheria from the bacteriological analysis of the Board of Health of New York city. Other observers think that in many cases it is not necessary, especially when the disease is epidemic; others believe it is necessary in every case. Koplick says that all cases of inflammation of the tonsil and throat, whether or no they are accompanied by the presence of the membrane, should be examined bacteriologically. Many cases of tonsillitis which cannot be distinguished grossly from simple non-diphtheritic tonsillitis are nevertheless true diphtheria, and the K.L. bacillus may be found in the plugs from these tonsils. Northrup thinks that although it is very desirable, our methods are not sufficiently accurate to certainly rely on the test at present. O'Dwyer says that too much importance should not be attached to the bacteriological examination in the present state of investigation. Out of 49 answers to this question 39 were affirmative. Dr. Buckingham says that the bacteriological examination is of great value, sometimes necessary, generally not.

(3.) The answer to the third question varies greatly. Some observers have apparently seen a large number of such cases; others have seen but few. Koplick thinks that these cases are extremely rare. Northrup has the same opinion. Williams says that at the hospitals they are about 20 per cent., and in private practice the percentage is larger. Booker says that when scarlet fever is epidemic in a city about one-fourth of the cases of pseudo-membranous anginas are not diphtheria. Morse, from observations made at the City Hospital, has carefully investigated over 20 cases of pseudo-membranous throats, in which the first examination did not show the K.L. bacillus, and in only 4 or 5 of these

was it not sooner or later found. In several cases the bacillus was found only after the disappearance of the membrane. He says, "I conclude from this that almost all cases which resemble diphtheria anatomically are due to the K.L. bacilli," and he explains the high percentage of cases without K.L. which have been reported to the fact that cultures have not been made carefully enough and often enough. "I also believe that many cases are hastily said to resemble diphtheria anatomically which on more careful examination would show not to do so. I am aware that my results have been vitiated by the patients being in the diphtheritic ward, where they may have added the bacilli to the pseudo-membranous throats not primarily due to the bacilli." In the pure scarlet-fever membranous throats Morse says that the K.L. bacillus is usually not present.

(4.) As to the fourth question, all observers agree that the simple membranous throats have a good prognosis. The cases recover when the membrane does not extend into the air-passages, but it may extend into the larynx and trachea, or death may take place from broncho-pneumonia. Dr. Buckingham thinks that the onset in these cases is more stormy.

(5.) In the fifth question of this, a mistake was made in the use of the word "infectious." Contagious should have been the word used. In answering most of the questions, answers are apparently based on the use of the word as meaning contagious, and the general opinion seems to be that such cases are capable of communication, although in a very much more limited degree than in the other.

With reference to the sixth question, the answers are generally negative. As a matter of fact, there can be no doubt that as far as transmission by air is concerned the hospital cannot serve as a focus of infection to the surrounding part of the city. There can be no doubt, however, that certain cases must be referred to infection from the hospital. It is of course difficult, if not impossible, to prove this. The wards of the hospital are an infected locality, and attendants, internes, and others going from these wards into houses elsewhere may convey the infection. Dr. Buckingham has never known a case of such transmission.

A CLINICAL STUDY OF CASES OF ANGINA RESEMBLING DIPHTHERIA IN WHICH THE BACILLI ARE REPORTED ABSENT.

By Dr. E. M. Buckingham.

Since the application of bacteriology to the study of diphtheria it has been questioned whether the cases in which the diphtheria (Klebs-Loeffler) bacilli are not found are really due to them or are cases of some other disease. There is nothing unreasonable in supposing them to be overlooked as is, we know, sometimes done, or that the membrane has outlasted the bacilli. It is also conceivable that there may be a disease of different etiology, but presenting identical appearances in the throat. It is, therefore, my purpose to study clinically the cases occurring in my last service in the diphtheria ward which were reported to be without these bacilli. In making this report I wish to express thanks to Dr. John L. Morse, who made many more examinations than the pathological department of the hospital had time for, who made clinical examinations of many of the cases, comparing notes with me afterward, and who in some instances visited patients' houses in search of information. No case referred to in this paper received the serum treatment, and all reported appearances in the mouth were recorded before the use of any local treatment.

There were a few cases in which bacilli were reported absent for a number of successive examinations, but in which they were afterward found. These may have become infected in the ward; but as we cannot know this to be true, none of them have been considered. There were twenty-four cases in which these bacilli were never found. Because of my then ignorance of the ease with which they may be overlooked, nine cases were insufficiently examined. These have been thrown aside and fifteen remain to be considered. No case is retained in which there was not an examination on entrance, a second within two days, and a third previous to discharge. The number of examinations in individual cases ranges from three to nine. Cultures were made from

more than one place at a single examination, as from both nostrils, both tonsils, the uvula, and the pharynx, but not all these regions were explored at a single examination. It is proposed to consider these cases with reference to temperature, vomiting, chill, pain, circulation, enlarged glands, coryza, condition of mouth, and length of stay in the hospital.

Perhaps it is not out of place to consider the normal temperature of diphtheria. Any kind of chart is sufficiently common in a diphtheria ward, but I believe that as a rule a high temperature means the presence of some complication. The temperature often rises with increasing dyspnœa. Patients occasionally have a certain rise, apparently from excitement when entering a hospital for any disease. Such a rise is generally of short duration. I have seen in a general ward temperatures of 102° and 103° in nervous children; but these were extreme cases. Slighter rises from this cause are common with children. All the diphtheria records in Vol. 18 of the contagious service and the first half of those in each Vol. 19 and Vol. 20 have been studied. All cases were thrown out if there was a record of sepsis, consolidated lung, cough, otorrhœa, operation on the throat, or other evident cause for fever. After this was done the charts of the accepted cases were studied. There were 67 such cases of uncomplicated diphtheria. In 22 the temperature did not reach 100°, and in 15 more did not exceed it. Including these there were 52 cases of the 67 in which the temperature did not exceed 101°, and 15 in which it did. It reached 102° in 13 cases only. In one of these the rise was apparently due to menstruation, while 5 were children in whom the second reading was 100° or lower, as were all subsequent readings. Of the remaining 7 cases, 1 died the day after admission. In 2 the brilliant red of the throat suggested scarlet fever, which was a common complication at the time, although these cases had no rash, and 4 times the temperature above 102° could be attributed to nothing but uncomplicated diphtheria. In 1 of these 4 the fall of 99 came on the second day and in the others sooner. It is possible that some cases with low temperature had a higher one at first, but that it fell before admission. I have a few

private records of cases in which the bacilli were detected in which there was a rise, the maximum temperature being 102°, and the fall immediate. Some cases, with other diseases, developing diphtheria in the hospital have had a rise of a few degrees with the initial symptoms, but of short duration; others have had no appreciable rise.

Among the 15 cases without bacilli, the temperature on admission was below 100° in 5 cases, did not exceed it in 7, did not exceed 101° in 9, and exceeded 102° in 4 cases. One of those below 100° was said to have had fever outside the hospital. There is no very remarkable difference in the percentage of these two series until we come to the higher groups. Of temperatures over 102° there were exactly as many in the one series of 15 cases as in the other of 67, after making allowance for probable causes of disturbance. These probable causes of disturbance did not exist in the smaller series. The high temperatures remained high for from 2½ to 4 days after admission, 6 to 10 days after initial symptoms.

The pulse is recorded as strong or good in 13 of the 15 cases, which is contrary to all my own experience in diphtheria, cases sick enough to be recognized having almost invariably weak, compressible pulses. Coryza existed in 5 cases; in 3 of them it was slight. Cervical glands were enlarged in 13 cases; in some slightly, in some considerably. In 2 of the 4 cases with highest temperature, the enlargement was slight, and in another it was doubtful. All cases had membrane; that is what sent them to the hospital; sometimes it was little, sometimes much. Tonsils and uvula were sometimes swollen, sometimes not; sometimes reddened, sometimes not. The tongue is reported red or coated with red edges in 6 cases, — nearly half the whole; but red is a comparative term, and while seeing the cases I had no suspicion of any such general redness. In 1 case, however, it is recorded as deep red, and in another as dark red with a thin film on the back. Redness is seldom mentioned in the records of the cases with bacilli, although their tongues are often described with some fulness. One throat is described as much inflamed, with little membrane; another, much swollen, with little membrane; another, much

injected, with little membrane; another, tonsils very much inflamed and swollen, with little membrane; another, throat injected with small patches on palate; yet another suggested follicular tonsillitis after the second day. Perhaps the rather general injection and swelling of the throat, together with little membrane in so many cases, and the presence of at least two highly reddened tongues, just suggest one of the acute exanthemata.

Vomiting occurred three times and nausea once, although but 3 of the patients were less than 16 years old. But one of the children vomited, and in her case, as with one of the adults, this symptom was severe. Too much importance should not be given to this symptom or to chill, headache, or other pain, each of which was present in 2 or more cases; because, although they are of trifling importance in most diphtherias, although they bear no comparison to the same symptoms in other diseases, yet careful questioning will show that they do sometimes exist to some degree.

One of the patients had kissed his child four or six days before admission, she having at the time diphtheria with intubation and scarlet fever with desquamation. Two patients were nurses in the scarlet-fever ward. One of them had never had scarlet fever, but had been in this ward for months. The other had been assigned to duty there but a few days before her sickness. Her previous history is unknown. At this time there were scarlet-fever patients in that ward with diphtheria bacilli in their throats. A fourth patient was taken sick on the same day with her sister, both having severe vomiting. The sister had very few bacilli on her first examination, although they increased rapidly.

One patient desquamated on the seventh day of the disease. She could have contracted scarlet fever in the ward where she had been five days, but if so it was without fever. In another, there was a suspicion of desquamation.

These fifteen cases of apparent diphtheria grouped about the single peculiarity of absent bacilli present as many high temperatures as more than four times as many cases in which the bacilli were found, and the temperature remained high for rather long periods. The heart was all but invariably unusually strong for a diphtheria heart.

There was on the whole an amount of redness and swelling in the mouth out of proportion to the membrane. Three of the fifteen patients are known to have been exposed to scarlet fever. Another case, otherwise one of the most suspicious with regard to temperature and vomiting, was taken sick on the same day as her sister, who had similar symptoms, and who had had very few bacilli at her first examination, this first examination being made in the diphtheria ward. One patient desquamated, he being otherwise one of the least suspicious in the group. In another patient, with much membrane and low temperature, there was a suspicion of desquamation.

Fifteen patients are a small number on which to formulate general rules. There is, however, perhaps some warrant for the strong suspicion, not certainty, that most of these cases were scarlet fever, with absence of or transitory rash. That the variations of scarlet fever are so innumerable adds force to the suspicion, as does the fact that that disease was very prevalent at the time. This suspicion was not entertained until the records were studied together, although I doubted these cases being diphtheria.

Three patients had traces of albumen in the urine. This is stated as a fact, not as throwing light on the question under discussion. All recovered. The longest stay in the hospital was thirty-six days. This patient was retained long after she would otherwise have been discharged, because of a certain pallor of the tonsil, which may have been due to treatment, but which is seen after sore throats of all descriptions and all sorts of treatment. The next longest stay was twenty-two days, for desquamation. The next longest, seventeen days, was in a woman who furnished nothing to the composite picture of scarlet fever unless a record of red tongue be such. She had five negative bacteriological examinations. Another patient with five examinations was discharged on the fourteenth day. The other eleven patients went out in from three to seven days after admission.

The cases analyzed are Vol. 18, pp. 174, 224, 250, 252. Vol. 19, pp. 4, 72. Vol. 20, pp. 8, 114, 130, 166, 190, 230, 268. Vol. 21, pp. 14, 34.

Some of the bacteriological examinations are recorded only at the laboratory.